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PART I.

Original Contributions.

ON SOME POINTS IN CONNEXION WITH THE TREATMENT OF SYMPATHETIC OPHTHALMIA,

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THE causes of sympathetic ophthalmia are now fairly well recognised; but how to avert the disease, and how to treat it when established, are still open questions.

Before considering these two points it may be well to recall the peculiar characters of the disease.

They may be summarised thus:—

1. The disease is induced in one eye by irritation set up in the other, generally either as the result of injury, or from degenerative changes going on in a damaged or lost eye.

The disease usually commences *without* pain as a warning, though it is commonly preceded by a certain amount of irritation, slight photophobia, impairment of accommodation sufficient to give rise to fatigue of the eye, and lacrimation.

One or all of these symptoms may be present, and in an adult would probably draw attention to the state of the eye, but in a child they are not, as a rule, sufficient to lead him to make complaint, so that in children the disease is not unfrequently far advanced before it is detected.

2. The inflammation is fibrinous or plastic, and, I believe, starts in the ciliary region, and extends simultaneously to the iris and choroid. I have never seen a sympathetically inflamed eye suppurate.

3. After a variable period the tension of the globe increases, and in this stage there is pain, which is sometimes very severe. If the disease be very acute, ciliary staphyloma will occur at this stage. Increased tension may continue for many months, and when staphyloma has commenced it will go on increasing. In the majority of cases, however, the staphyloma does not occur, and after a variable time the excessive tension subsides, then becomes normal, and ultimately the eye softens. The eye is then irreparably lost.

I. *How to avert the Disease.*—My own experience leads me to believe that if an eye have received a severe injury, and it be excised before any symptoms of irritation occur in the opposite eye, then the sound eye is safe from sympathetic ophthalmia.

There may be a few apparent exceptions to this rule, but it has not yet been my misfortune to meet them. I have seen cases in which there has been very slight irritation only in the uninjured eye, and for this the injured eye has been removed. After the operation the irritation has completely subsided, and the safety of the eye has apparently been secured. But after a lapse of time varying from a few weeks or months to many years, the irritation has recurred, and rapidly drifted into the true sympathetic irido-choroiditis, ending in total loss of the eye. These cases can, however, hardly be considered as exceptions to the general rule which I have laid down. Sympathetic irritation is closely allied to sympathetic ophthalmia, and although it presents features different from true sympathetic inflammation, yet it may and frequently does drift into it, as rodent ulcer, which in its early stages is distinct from epithelioma, may at a later date pass into and assume all the characters of this formidable disease.

Now one of the peculiarities of sympathetic ophthalmia is

that it is recurrent, as indeed are all the forms of iritis, no matter how induced; and sympathetic irritation shares also this characteristic, that it is recurrent, and an eye which has once been subjected to sympathetic irritation may at some future period have a recurrence which may drift into well marked sympathetic ophthalmia. Such I think is the explanation of the two following cases :—

CASE I.—*Sympathetic Irritation coming on Seven Months after an Injury—Injured Eye removed—Complete Recovery and Good Sight for nearly Nine Years—Recurrence of Inflammation—Loss of Sight—Staphyloma—Sclerotomy.*

Robert W., æt. 33, was admitted into the Royal London Ophthalmic Hospital on June 6, 1880. Ten years before, he received an injury to the right eye from a blow with a small piece of wood, which from his description appeared to have ruptured the globe. Seven months after the injury he had sympathetic irritation in the left eye, and for this the right eye was excised. All irritation ceased after the operation, and the recovery was complete. He was sure that he could see with the eye "as well as he ever did in his life." He could see the smallest type, or the markings on the minute-hand dial of a watch. For nearly nine years he continued quite well. About twelve months before admission he had slight pain in the eye with failure of sight, and this increased until he applied at Moorfields Hospital, when he was admitted as an in-patient.

On admission the eye presented all the well marked appearances of advanced sympathetic ophthalmia. There was complete posterior synechia; the pupillary space was filled with opaque membrane; the iris, and especially its peripheral portion, was bulged towards the cornea, so that the anterior chamber was almost obliterated. There was a general bulging of the ciliary region, with a thinning of the sclerotic, and at the upper and inner part there was a large irregular staphylomatous protrusion. The tension of the eye was but a little above normal. The man could not count fingers, and had only perception of light in the outer part of the field. He had severe constant pain in the eyeball, which kept him awake at night.

In this patient the disease may be said to have run its course. His sight was reduced to a mere perception and he had staphyloma; but in addition he had pain, and it was to the relief of the pain that the treatment was now directed.

On June 18 sclerotomy was performed. A thin Graefe's cataract knife was made to puncture the sclerotic in the upper region of the eyeball, close to the corneo-sclerotic junction, and carried across the anterior chamber in front of the iris, the counter-puncture being made at a corresponding point on the opposite side, and the knife withdrawn. So shallow was the anterior chamber that a few of the fibres of the iris were caught by the point of the knife as it traversed it.

The result of this operation was very satisfactory. On June 29 the staphyloma had quite subsided, and the eye was quite free from pain. The sight was not materially improved. The eye was soft, —T. 1.

Case II.—*Sympathetic Ophthalmia coming on Four Weeks after an Injury to the Opposite Eye—Excision—Complete Recovery—Recurrence of Inflammation—Loss of Sight.*

Fred. H., æt. 9, was admitted into the Royal London Ophthalmic Hospital on the 23rd of January, 1880, four days after receiving the following injury. Whilst playing, he was struck on the left eye with a piece of stick which was thrown at him. On admission there was a small vertical rent on the inner side, but within the margin of the cornea, through which the iris was prolapsed, and there was traumatic cataract. The prolapsed iris was snipped off. Between the fourth and fifth week symptoms of irritation came on in the right eye, and the injured eye was at once excised. After the operation all the symptoms of irritation subsided, and the eye apparently completely recovered. The child could see the smallest type, and had good distant vision.

On April 2 the lad was brought to the Hospital complaining of a slight recurrence of irritation in the eye. There was some lacrimation and a little ciliary injection. These symptoms increased in spite of treatment.

On May 7 the boy was admitted into the Hospital, where he

continued until the end of June. There was at this time complete posterior synechia, lymph in the pupillary space, and a very shallow anterior chamber. The eye was quiet, and could count fingers at six feet, and read letters of Jaeger 16 and 18.

II. *How to proceed—(a) when the Injured Eye is totally Blind; (b) when the Injured Eye still retains some Sight.*—An eye has received an injury, and sympathetic ophthalmia has occurred in the other eye: Should the injured eye be removed? Experience has convinced me that the removal of the injured eye when sympathetic ophthalmia is established in the other eye will not arrest the disease; the inflammation will go on as if the source of the primary irritation remained. Nevertheless, if the injured eye be hopelessly blind, it should, I think, be excised, in order to give the patient the chance of any benefit that might accrue therefrom.

The removal of the injured eye may arrest sympathetic irritation, and frequently does, but it will not stay the progress of sympathetic ophthalmia. It is, therefore, doubtful whether any good will ensue from the removal of the injured eye. On several occasions, where the injured eye has retained some sight, I have seen the opposite eye destroyed by sympathetic ophthalmia, while the injured eye has ultimately so recovered that useful sight has been restored, and the patient has been able to get about without assistance. The conclusion at which I have arrived from watching these cases is, therefore, that if sympathetic ophthalmia be established, the injured eye should not be removed if it retains any sight.

III. *How to treat the Disease.*—In the curative treatment of sympathetic ophthalmia we have much to learn. At present we can do but little, as we have no means by which we can control the disease with certainty. The mode of treatment which seems to benefit in one case fails to afford any relief in another. On one point I feel quite satisfied: that operative interference in the inflamed eye is positively prejudicial in the early stages of sympathetic ophthalmia. I feel also equally certain that in the later stages when the pupil is

closed and false membrane fills the pupillary space, that no operation should be done except when the eye is absolutely quiet, and there is no inflammatory action.

My present feeling is that *in the early stage* of the disease the eye should be excluded from all light. The darkest possible glasses should be worn out-of-doors, and whilst at home the patient should be in a dark room with a black bandage across the eyes, provided the bandage does not produce discomfort by occasioning heat and conjunctival secretion from the lids. The aim is to give the eye as perfect rest as possible. During this stage mercurial inunction into the temple every night may be tried and continued for two or three months, care being taken to avoid salivation by diminishing or omitting the ointment for a few days as required. At the same time quinine in from half grain to two grain doses, according to the age of the patient, combined with small doses of the extract of belladonna, may be given two or three times a day. A weak solution (0·25 per cent.) of atropine or daturine may be dropped once a day into the eye, but in the later stage of the disease, when the whole posterior surface of the iris is adherent to the capsule of the lens, I believe that all mydriatics do harm by stimulating the dilating fibres of the iris to contract when from the adhesion of the iris to the lens-capsule they cannot act. Mydriatics also favour the development of the state of increased tension which usually comes on at a later period of the disease.

In *the second stage* of the disease when the union between the iris and lens-capsule has been effected, but after the acute symptoms have subsided and the eye is quiet, it is a question whether an operation may be attempted to improve the sight. My opinion is that if the sight is sufficient for immediate requirements, and will enable the patient to walk about without assistance, the eye should be left alone. It is the only eye, and operations on eyes sympathetically inflamed are so unfavourable that it is better to let the patient enjoy the sight he has, rather than to risk the loss of it with the prospect only of a slight improvement.

If, however, the sight is so defective as to be almost useless, and there is a fair field of vision, then an attempt should be made to improve it by an operation. I have for a long time felt satisfied that the great object to be attained in these cases is to get rid of the lens, as I have noticed that when this has been satisfactorily accomplished the tendency to the recurrences of inflammation is greatly diminished, and an artificial pupil may be afterwards made, through which the patient will enjoy greatly improved sight. The removal of the lens is an operation of difficulty. It may be accomplished thus:—First make a section of the cornea with a Graefe's cataract knife or a broad keratome, then tear open the pupil and through the capsule of lens with a cystotome, or if this be insufficient, remove a portion of iris and the adherent lens-capsule with a pair of iris scissors, and, lastly, with a little pressure on the globe with the back of the curette cause the escape of the lens through the corneal wound. This operation may generally be accomplished without the loss of any vitreous, and the eye will usually recover from its effects well, but the pupil will probably again become closed. Another operation will afterwards be required for the formation of a new pupil.

The third stage of the disease is characterised by increased tension of the globe, and this condition is generally associated with pain, oftentimes very severe, and sufficient to make the patient willing to submit to any means suggested for his relief. This state of tension lasts a variable period, but, as a rule, it ultimately subsides, and the eye softens. If the state of tension be long continued the little sight that is left soon vanishes. When the eye has reached this stage of increased tension active treatment is necessary. An attempt to do an iridectomy I have always found unsuccessful. The iris is adherent throughout its posterior surface to the lens-capsule, and is either so rotten that the forceps pass through it, and cannot seize a piece to draw it out, or else the iris has become tough and fibrous, and simply tears like calico under the grip of the forceps, and no portion of it can be satisfactorily removed.

During the last eight months I have performed the operation of sclerotomy in these cases, and have been pleased with the result. The knife has been made to pierce the sclerotic just external to the corneo-sclerotic junction, rather higher than the point selected for a Graefe's extraction of cataract, and carried in front of the iris; the counter-puncture has been made at a corresponding point on the opposite side. After slight cutting, so as to make the section a little broader than the breadth of the blade, the knife has been withdrawn, leaving a wide bridge between the sections.

In each instance the pain has been relieved immediately, and in two (Cases I and III), in which there were ciliary staphylomata, the bulgings subsided, and the eyes became soft. In one patient (Case III), in whom the pain was so excessive, that morphia had to be injected subcutaneously twice a day, the relief was so rapid that after the operation the patient slept without the morphia injections, which were not again required. In this case the sight also was improved. Before the operation the man could only just count fingers when held close to the face, and when he left the hospital he could read Jaeger 19 at 2 feet.

Case III.—*Sympathetic Ophthalmia coming on about ten weeks after the Injury—Loss of Sight—Increased Tension—Ciliary Staphyloma—Sclerotomy—Good result.*

Edward T., a porter, æt. 31, was first admitted into the Ophthalmic Hospital on August 8, 1879. His right eye had been injured by the explosion of a soda-water bottle in the previous May, and the sight of the left eye began to fail about seven weeks afterwards.

On admission there was a puckered cicatrix in the ciliary region of the right eye, and the globe was shrunken. In the left eye there was a slight ciliary blush. The pupil was very sluggish, almost inactive, and a thin film of lymph occupied the pupillary space. There were minute dots of lymph on the posterior surface of the lower half of cornea. This eye could only read Jaeger 16. Tension was normal.

The injured eye was excised. The patient remained under

treatment until September 22, when he was discharged. The eye was quiet, and had so much improved that he could read Jaeger 2 at 10″ and V = $\frac{20}{40}$.

On November 7, 1879, the patient was readmitted into the Hospital with a recurrence of the sympathetic inflammation of the left eye. There was complete posterior synechia, and the pupillary space was filled with opaque membrane.

The vision was very bad. He could hardly see fingers. He was discharged on the 29th. The eye much quieter and less injected. Sight not improved.

On December 12, 1879, the patient was again readmitted. Soon after returning home he had had another attack of inflammation of the eye, accompanied with pain, which was relieved by leeches and blisters to the temple. The pupil was on readmission excluded and occluded, and the iris was bulging. T. 1.

On December 28 he had great pain; a sense of burning in the forehead and eyeball; much ciliary injection, lacrimation, and photophobia. T. 2. Ordered two leeches to temple.

From this time the symptoms increased in severity. The pain became so severe that relief could only be obtained by subcutaneous injections of morphia, given always once, and frequently twice a day.

Other sedatives were tried, but none gave the relief of the hypodermic injections of morphia.

The tension of the globe continued T. 2. Drops of sulphate of eserine were tried, but without influencing the tension. The sclerotic in the upper and outer half of the ciliary region began to thin, and slightly bulge. The sight was so reduced that he could only count fingers held very close to the face.

February 13 the patient was placed under ether, and a sclerotomy was made upwards, a good bridge of sclerotic being left. The relief from the operation was immediate. The patient passed a good night without the hypodermic injection of morphia, and next morning the tension was normal.

On the 24th the iris was still bulging, but there was no pain. The patient could read Jaeger 19 at 2 feet.

On March 3 he was discharged. The tension of the globe had slightly increased. He was free from pain, and he could read letters of Jaeger 16.

ON A THEORY OF GLAUCOMA.

By W. A. Brailey, M.D.

By the term glaucoma, I mean not only primary glaucoma inclusive of its hæmorrhagic form, but also the cases where increased tension is secondary to injury, corneal ulcer, tumours, or to various inflammations of the uveal tract.

The eye is a capsule of fluid, with walls extensible in youth, but becoming more rigid as age advances. The fluid in it, like the tissues of the body generally, is in a state of constant change. Its ingress probably varies somewhat in health with the conditions of the circulation. The channels of egress are doubtless sufficient for amounts varying within considerable limits.

But, when compared with other parts of the body, for example, areolar tissue, the capacity of the eye for accommodating an excess of fluid is limited. Moreover, the tissues liable to compression here are both delicate and important.

An accumulation of fluid, whether due to an inflow beyond what can be carried off by the normal channels of outflow, or to obstructed outflow, or to both causes acting together, constitutes the condition of increased tension called glaucoma.

The theory of an increased inflow has had Donders as its distinguished exponent, by whom it was attributed to a neurosis.

That of obstructed outflow has been maintained by Knies, Weber, Wecker, and others. These agree generally in putting the principal seat of obstruction at Schlemm's canal, whether by its inflammation or by the apposition of the iris to its entrance. Their differences mostly concern the way in which this apposition has been effected; some attributing it to the swelling of the ciliary folds, others to the advance or an alteration in shape of the lens. In the opinion

of the author both principal theories have their value, and each is capable even by itself of accounting for tension.

He attributes increased inflow sometimes to a visibly increased blood supply, and sometimes to inflammation, especially of the ciliary body and iris. Diminished outflow he ascribes, in most cases, to an obstruction in the region of the ligamentum pectinatum, and in some few, in the exits from the perichoroidal space. He finds, however, in most cases such a condition of the optic nerve as would hinder any passage of fluids along it; though that this is a channel of exit in the normal eye is unproved by experiment. Obstruction at the entrance to Schlemm's canal, he ascribes in most cases to valvular action of the iris, induced either by its position or by the condition of it, and the ciliary body.

The fluids in the eye may be divided for convenience into three systems:—

A. That of the vitreous and aqueous chambers, inclusive of any found in the subhyaloid space.
B. That of the retina.
C. That of the choroid.

The two last of these are constantly recruiting the fluids of the first, and any accumulation in them is rare. On this account they have not often much direct influence on the tension. They will be described hereafter, and their influence discussed.

The fluids contained in the meshes of the vitreous and in the aqueous chambers are in pretty free communication across the so-called canal of Petit external to the lens margin, and through the pupillary aperture. They are recruited by the transudation which has taken place from the blood-vessels for the nourishment of the tissues, more especially from those of the uveal tract, as containing by far the greatest bulk of blood within the eye. And of this tract the iris and ciliary body are, perhaps, the most important parts, as being immediately in contact with the vitreous; whereas the choroid is separated from it by the retina, which must offer at least some obstruc-

tion to the passage of fluid through it. Some conditions of detachment of retina appear to point to this: and some conditions of cysts of the iris afford indications that considerable fluid escapes from its uveal surface. Fluid from the proper retinal blood-vessels without doubt drains into the vitreous cavity, but its amount is comparatively small, as they are far less in size and number than those of the uveal tract.

In the body generally it has been shown that the amount of transudation varies with the blood-pressure. This pressure is increased locally whenever any artery is dilated up to its origin from a larger trunk. The pressure in its capillaries becomes also increased beyond the normal, and an increased escape of fluid results.

This enlargement of the vessels is seen *within* the eye in numerous cases (see Table, page 94). It affects almost solely the blood-vessels of the ciliary region and iris. *Outside* the eye the vessels forming part of the supply of this region, the anterior ciliary arteries, are seen during life enlarged in many cases in their course beneath the conjunctiva.

This enlargement is probably the consequence of an affection of the vaso-motor nerves, and a *neurosis* theory will explain the occasional association of glaucoma with mental anxiety and with neuralgia. Indeed, it has been established by experiment that the 5th and sympathetic nerves have an influence on the tension. The speedy onset and occasional spontaneous subsidence of glaucoma will also be thus explained.

The arterial dilatation may be independent of visible external causes, or it may arise in consequence of a blow or wound, and constitute by itself the cause of a glaucoma (see Group III, page 114). Its existence is by no means necessary as a cause of tension, though it is found in many cases of primary, and still more frequently in hæmorrhagic glaucoma. I think its existence may be due in some of these cases to irritation of the nerves of the ciliary body consequent on the extreme stretching of the fibres of the suspensory apparatus of the lens. The mode in which this stretching

is set up in consequence of atrophy and alteration of shape of the ciliary body will be subsequently explained. The same explanation will apply to the occasional extremely rapid occurrence of tension after the lens has been partially or entirely displaced backwards by violence, and sometimes also to cases where glaucoma has quickly followed the forcible breaking up of cataracts by needling.

I infer generally that arterial enlargement if not found at excision has never been in existence. It is possible, however, that if it have not been of so long duration that the usually occurring atrophy of the arterial walls have not supervened, it may have existed, and then afterwards subsided. Such a suggestion would explain the phenomena of intermittent glaucoma, and the occasional cure of glaucoma by paracentesis alone.

An inflammation of the ciliary body and iris of a kind ultimately passing into atrophy or into atrophy with sclerosis, has been described in another paper* as frequently existing especially in young subjects with disease of short duration. The opinion has been expressed there, that some inflammation always precedes the atrophy, that it exists prior to the establishment of the tension, and that in most cases of Primary Glaucoma, especially in old persons, its degree is but slight.

In such case it will have little effect in increasing the fluids of the aqueous and vitreous chambers, and any excess derived from it will be easily carried off by the natural channels. It will probably be different if the inflammation be acute, and here we may presume a hypersecretion which will constitute a material addition.

But when sclerosis and atrophy are established, there will be rather an actual diminution of the fluids normally recruiting the vitreous and aqueous, unless an enlarged arterial supply exist.

It has been experimentally established that the excess fluids of the vitreous and aqueous chambers have their normal exits from the anterior chamber through the endothe-

* "On the Condition of the Optic Nerve, Ciliary Body, and Iris, in Increased Tension," page 86.

lium-covered meshwork of the ligamentum pectinatum, after passing from the vitreous chamber, where they are probably derived from the choroidal and retinal vessels, through the canal of Petit, where they are recruited from the ciliary folds, into the posterior chamber of the aqueous humour, where is added to them the fluid escaped from the uveal pigment stratum of the iris, and through the pupillary aperture.

In consequence of this current, which is certainly very slow in health, there is normally some slight excess of pressure behind the iris over that in the anterior chamber, though this power is by no means sufficient to move the thick and contractile diaphragm formed by the normal iris.

It has, however, been established that a very slight increase of pressure in the vitreous over that in the aqueous chamber, is sufficient to carry forwards in the eyes of recently killed pigs the periphery of the iris, and block further exit through the ligamentum pectinatum.*

If such a movement ever occur in living human eyes, it is probably of extremely rare occurrence with unaltered ciliary body and iris.† For example, it has not taken place even in Case 352, nor in 101 of Group III. Some cases, however, seem to show that it may be induced without any increased current of fluid, provided that the periphery of the anterior chamber has been so narrowed in an antero-posterior direction as to cause a difficulty in the passage of fluid between the iris and cornea towards the ligamentum pectinatum. For then the pressure on the front surface of the peripheral part of the iris, will tend to become decidedly less than that on its posterior surface. This condition of things is brought about by the atrophy of the ciliary body as elsewhere explained (page 86). Under these circumstances, it is quite intelligible that a very slight additional atrophy at any one point will accentuate the difference of pressure and carry forwards the iris there. The pressure on the front surface of

* Priestley Smith "On Glaucoma," p. 133.

† It is obvious that during life the conditions of the experiments just referred to are only fulfilled when the cornea is perforated.

the immediately adjacent parts of the iris will then also be diminished, and thus the application of the iris periphery will spread laterally till its entire circuit is involved, or at least as much of it as is implicated in the atrophy of the ciliary body.*

There are, however, other cases with applied iris in which the atrophy of the ciliary body, though present in a certain degree, obviously could not have advanced the periphery of the latter so much as to have induced its valvular action without increased pressure behind it.

For in these the iris is applied at a point distinctly in advance of its base, which may even be separated from the front wall of the extreme periphery of the chamber by a little fluid imprisoned there (see page 91). This condition of things can only happen when the chamber is extended backwards by the atrophy and alteration in shape of the ciliary body.

I attribute the position of the iris in these cases to an increased flow of fluid towards Schlemm's canal from the ciliary body, due either to its inflammation or to the increased vascular supply already referred to. The valvular action of the iris is further facilitated by its atrophy, which is always developed in connexion with the inflammation or atrophy of the ciliary body, and by any shallowness of the anterior chamber. The disposition to it is increased if there have been anterior synechia, or if the eye be hypermetropic.

It is perfectly clear that in cases of perforated corneal ulcer the iris acts as a valve. For whether, as is far more usual, it line the entire posterior corneal surface, or be applied only partially as explained in another paper, its effect is to prevent exit of the fluid from behind it.

Its application is due to the sudden diminution of pressure on its anterior surface, and so long as any leakage continues through the region of the ulcer, the anterior chamber cannot be re-established. If, however, the perforation be so limited that it can be completely plugged by the iris and

* The Ligamentum Pectinatum is by its shrinking the especial cause of the approximation of the iris periphery to the cornea (see pages 92, 93).

thus rapidly closed, the anterior chamber may be retained, and the eye be left with only a point-like anterior synechia. If the perforation be large, the iris, if it has not, when the leakage ceases, attained to the atrophic condition to which it is rapidly progressing, may, by the secretion of fluid from its anterior surface, partly re-establish the anterior chamber.

The failure of this secretion in advanced cases of glaucoma is the reason why the lens and iris sometimes remain jammed up against the cornea after operations, giving rise to the condition known as Malignant Glaucoma.

If the entrance to the canal of Schlemm be blocked by inflammation, as appears to be the case in "Iritis Serosa," the pressure on the two sides of the iris will be equal, and it will have no tendency to advance even though the tension be extreme.

It is obvious that exclusion or occlusion of the pupil will also prevent escape of fluids from behind the iris through the canal of Schlemm and thus cause glaucoma.

A rapid increase of the contents of the eye, as in suppurative panophthalmitis, will also cause increased tension, while one slowly progressing as in the case of many tumours, will be compensated for by atrophy of neighbouring parts, the vitreous for example, unless a channel of exit be obstructed by its growth.

With regard to the fluids of the retina, their source and mode of exit are more matters of speculation, being rather inferred from pathological conditions than determined directly by experiment.

The positions in which leucocytes are found in some diseases (see page 138), appear to show that fluid from the proper retinal blood-vessels drains through the internal retinal surface. Some may also find its way along the sheaths of the retinal vessels into those of the central vessels of the nerve.

The disease of the retinal vessels which exists in all cases of Hæmorrhagic Glaucoma, and also in most other cases of Primary Glaucoma, is fully described later (see page 133).

It has no bearing on the tension, for a similar condition of the vascular walls without tension is found in the eyes as well as in other organs of many patients who are the subjects of various general diseases.

Moreover, there is evidence that in Glaucoma it may either exist previous to or be developed subsequent to the tension (see Case 483).

The fluid of the outer retinal layers is undoubtedly derived from the chorio-capillaris. It probably finds its principal exit towards the vitreous cavity, thus completely traversing the retina in its course. Some, however, appears to escape by means of the optic nerve, either along the sheaths of the central vessels in company with that from the internal retinal layers as previously described, or along the lymph-spaces of the fibrous stroma of the nerve. The function of the optic nerve as a channel of exit is, however, as yet unproved, but if existing it would be interfered with by the sclerosis of the lamina cribrosa and nerve elsewhere described as existing in Glaucoma (see page 87).

There are, sometimes, in this disease indications of an accumulation in the inter-granule layer near the optic disc, and more often in the neighbourhood of the ora serrata. Besides this, there are very often similar indications of an accumulation in front of the lamina cribrosa, and there is, sometimes, traceable a fissure or layer of looser tissue leading to this from the inter-granule layer. Where the retina is detached the stratum of loose œdematous tissue appears to communicate with the sub-retinal space.

These appearances would be consistent with the existence of a current from the outer retinal layers to the lymph channels of the optic nerve.

In Glaucoma the retina is sometimes found extremely cystic, corresponding to where the sclerotic is staphylomatous over a small area of inflamed and thinned choroid. The explanation of this is not so clear, though the same ideas as to the escape of fluid from the chorio-capillaris might be applicable here.

The relative quantity and effects of the fluids derived from the retinal blood-vessels must in any case be small. Moreover, there are never indications that the retinal blood-supply is increased in Primary Glaucoma, while it is certainly diminished when the tension has been fully established.

And if sclerosis of the nerve and lamina cribrosa be a means of retaining some of the waste fluids of the retina within the globe, it has, when unaided, no effect on the tension, for it exists together with normal tension in many cases of cerebral disease.

The Choroid derives its fluid from its own blood-vessels, and has probably one of its outflows into the retina as above stated.

But some fluid, especially from its outer layers, escapes by way of the perichoroidal lymph space into the cavity of the capsule of Tenon along the spaces surrounding the venæ vorticosæ and the long ciliary arteries and nerves.

There is no reason to suppose this outflow interfered with in any of the cases given in the Table (p. 94), but there is evidence of increased fluid in the perichoroidal space in some few (see Nos. 214 and 274).

This fluid would be increased not only from inflammation of the choroid attended by hypersecretion as in choroiditis serosa, but from obstructed efflux through tumours involving or pressing on the outlets.

It is difficult to judge of the effects of choroiditis serosa alone, as it rarely, if ever, occurs without iritis and cyclitis, the secretion from which has its exit by the canal of Schlemm.

It will be seen that none of the cases given in the Table are without one or more of the conditions of diminished efflux or increased afflux to which we have attributed increased tension.

Out of the entire number of cases reported in the Table, two have sclerosis of optic nerve only. (It is coupled, however, in each case with disseminated choroiditis.)

Forty have access to the canal of Schlemm obstructed, and inflammation or sclerosis of the optic nerve.

Four have only enlarged arteries in the ciliary region, and inflammation or sclerosis of optic nerve.

Thirty-eight have enlarged arteries in the ciliary region, access to canal of Schlemm obstructed, together with inflammation or sclerosis of the optic nerve.

One has access to the canal of Schlemm obstructed by an universal posterior synechia, as well as a little enlargement of the arteries, together with inflammation and sclerosis of the optic nerve.

Three (Group VI) have iris periphery free, no atrophy of it or of ciliary body, some neuritis, also inflammation of iris, ciliary body and choroid, with evidence of serous hypersecretion.

Testing this short account of the principal agents concerned in the theory by the Table of cases given at page 94, we find that whenever the canal of Schlemm is blocked by the iris, this condition is accompanied either by perforated corneal ulcer or by an acute inflammation of the ciliary body, with little or no atrophy, or more commonly by an extreme atrophy of the same with or without sclerosis. Any of these conditions may be accompanied by enlarged arteries.

In Group I (Primary Glaucoma) we find the canal of Schlemm closed, or access to it extremely narrowed in 93 per cent. of the cases. Access to it is free in 7 per cent. The ciliary arteries are enlarged in 50 per cent. The canal of Schlemm is obstructed without arterial change in 12 out of the 29 cases.

It is thus clear that obstruction at the canal of Schlemm is sufficient to maintain a glaucomatous condition, and even to originate one, provided that the iris have been brought into the required position.

There are in this Group two cases (Nos. 406 and 265) exceptional as regards the canal of Schlemm and the iris adhesion. Nor do we even find in them sufficient inflammation of the ciliary body to warrant us in suggesting this as the cause of the tension. They will, however, both be seen to

have sclerosis of the optic nerve, and in the first there is also a distinct inflammatory affection of the choroid. Being thus qualified, and constituting such a minority of the cases in the Table, I shall not further discuss them here, but leave my readers to judge their value in relation to the theory suggested, as their principal pathological conditions are fully reported in the Table.

In Group II (Hæmorrhagic Glaucoma) access to the canal of Schlemm is obstructed in every case, and the arteries are enlarged in a greater proportion than in Group I. Thus we should expect to find the Glaucoma more acutely developed here, which is in fact the case. Moreover, the atrophy and sclerosis are also greater than in Group I. No exceptional case is found here.

Group III (Glaucoma after accidents), which by the conditions under which it is formed contains cases of short duration, with little inflammation at the seat of injury, has consequently usually but little inflammation or sclerosis of the ciliary body. Therefore, we are prepared to find the iris periphery not applied in some cases. The enlarged arteries, on the other hand, are of far more frequent occurrence than in the other groups, being present in 87 per cent.

There appears to be one partially exceptional case, No. 214, but in it the iris is applied at one part, and there is present, as in one of the exceptional cases of Group I, some disseminated choroiditis.

In Group IV (Corneal ulcers) the canal of Schlemm is blocked in every case, but sclerosis and enlarged arteries are decidedly more rare. The muscle is always somewhat inflamed at an early stage, and later tends to atrophy. If an adult or old person be the subject of the ulcer, there may also be sclerosis. The optic nerve is always affected. Neuritis with an inflammation of the tissue between the innermost and outermost nerve-sheaths, makes its appearance very speedily after the development of the corneal affection, and before the establishment of the tension. It generally passes on to sclerosis.

Group V (Buphthalmos) is the same as Group IV, insomuch that access to the canal of Schlemm is blocked in every case, and also in other respects.

In Group VI (Iritis serosa) the ligamentum pectinatum and neighbourhood of the canal of Schlemm are inflamed in every case. There is much inflammation of the ciliary body and iris, but no atrophy. The iris periphery is widely removed from the cornea, except where in cases 285 and 509, it has become partly entangled in the iridectomy wound.

Most theories of Glaucoma hitherto advanced rest on an obstructed outflow through the ligamentum pectinatum either by an inflammation or by apposition of the peripheral part of the iris. Their differences consist in the mode in which the iris is supposed to acquire this position.

Some attribute it to an advance of the lens from increased pressure from behind it.

But it appears to be almost impossible that any advance of the lens could produce this effect. For in my microscopic specimens where the lens capsule is left with its normal attachment to the ciliary body, its periphery is on a level considerably internal to the canal of Schlemm. And the extreme angle of the anterior chamber, the point where in many cases with great atrophy of the ciliary body the adhesion appears to begin, is normally even a trifle more external than Schlemm's canal.

And in the pseudo-glioma often seen in young children, while the centre of the lens is distinctly advanced very near to the cornea, the peripheral part of the anterior chamber is deepened by the drawing back of the most circumferential part of the iris.

Such a condition is also not uncommon in adults in whose vitreous chamber organised inflammatory products have advanced the lens, while retracting the iris periphery.

An extreme advance of the lens, as after paracentesis of the cornea, would prevent access to the canal of Schlemm by pressing the *pupillary* part of the lens against the cornea.

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Then the iris also would apply itself and prevent further approach to the canal of Schlemm. But this certainly does not take place in the course of a Glaucoma.

It is more plausible that the ciliary folds swollen by vascular injection could have advanced the periphery of the iris. Only a few of my specimens, however, give any support to this explanation. Moreover, the swollen folds should have produced their effect also in iritis and cyclitis serosa, and in cases of Glaucoma with unusually deep anterior chamber. The theory fails also to explain the absence of advance of the iris in some cases of Group III.

Others place the obstructed exit at the canal of Petit by an increased diameter of the lens occurring with age. No measurements, as far as I am aware, are extant showing the increase of the lens upon which this theory is based. Moreover, one would *à priori* expect a shrinking in this direction coincident with the antero-posterior diminution that undoubtedly takes place.

Such an atrophy of the ciliary body as has been described, if occurring before the closure of the canal of Schlemm, would widen the space surrounding the lens margin. Moreover, this theory will not explain the occurrence of Glaucoma in young subjects, nor the frequent rapid development of tension in an eye whose lens has been dislocated into the vitreous chamber by a blow (see Cases 101 and 421) nor the occurrence of Glaucoma after cataract extraction without entanglement of the iris or occlusion of the pupil.

Every theory of Glaucoma though based on howsoever many examinations of eyes excised on account of severe disease, is open to the objection that the changes upon which it rests may have arisen subsequently to the establishment of the tension. It would require *post-mortem* examinations of very early cases in order to show that inflammation, atrophy, and sclerosis, whether of muscle or nerve, have been in existence previously. And this good fortune does not fall to the lot of everyone. Schnabel, however, a most competent observer, in referring to the author's previous paper on

Atrophy of the Ciliary Muscle, records* the pathological examination of a case in which he found the atrophy of the ciliary muscle distinctly developed with tension of only a few hours' duration.

Moreover, both atrophy and sclerosis are fully established in some cases of a few months' history only (see Group I, Cases 241 and 763).

On the other hand, some cases of injury (see Group III) prove conclusively that atrophy and sclerosis are developed with extreme rapidity.

But again, many phenomena are seen in eyes about to become glaucomatous before any increase of tension can be clinically detected. Of these, the change in refraction, the diminution in accommodative power, and the sluggish pupil would be explained by the atrophic changes in the shape and texture of the ciliary body and iris, whether the result of a slow inflammation imperceptible to the eye of the surgeon, or non-inflammatory.

The occurrence of Glaucoma without such premonitory symptoms would conform with the enlargement of arterial supply, and, perhaps, even with the establishment of an acute inflammation. I have before suggested that the inflammation of the ciliary body and iris is attended with increased fluid. It is of a kind leading rapidly to atrophy, which is even more conspicuously seen in the iris than in the ciliary body. The microscopical appearances in my specimens conform more frequently with the hypothesis of fluid pressure than with any other.

Reasons have been before given why the iris base should not, in normal conditions, act as a valve. But the case is altered when with an atrophied iris, more especially one whose base is already somewhat approximated to the cornea by ciliary atrophy, we have an increased escape of fluid from behind it.

With these three circumstances combined, it is possible to conceive the iris base advancing under pressure of the current of fluid.

* Knapp and Moos, Archives of Ophthalmology, vol. vii, p. 279.

An extremely slight inflammation of the ciliary body would not be attended with sufficient hypersecretion to account for the movement of the iris, but the atrophy subsequent to it would so approximate the iris to the cornea, that it would be under the influence of the normal or even slightly diminished flow of fluid towards Schlemm's canal.

A FURTHER INVESTIGATION OF THE PATHOLOGY OF GLAUCOMA.

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In an essay on glaucoma,* published in October last, I advanced a theory which was to a certain extent new. It was founded in part upon the discoveries of Leber, Knies, and Weber, in part upon the results of certain experiments of my own. It was supported by a good deal of circumstantial evidence drawn from the clinical aspects of the disease; but it was at the time of writing a theory and nothing more—proof of its truth was wanting. As the result of further investigation, I am now able to offer some additional evidence in connexion with certain of the doubtful points.

A brief re-statement of the steps by which the theory was arrived at, and of the theory itself, will be convenient here.

a. The intraocular fluid escapes from the interior of the eye, at the angle of the anterior chamber (Leber).

b. In glaucomatous eyes microscopically examined, this angle is found to be narrowed or closed. In the great majority of cases a firm adhesion exists between the periphery of the iris and the opposing surface of the cornea (Knies, Weber).

c. It is manifest that this closure is connected with the undue retention of the intraocular fluid, and the excess of tension, which characterise glaucoma, especially when we remember that the only operative procedures which have a definitely curative action on glaucoma are practised at the angle of the anterior chamber.

d. How does the closure of the angle of the anterior

* "Glaucoma: its Causes, Symptoms, Pathology, and Treatment." (The Jacksonian Essay for 1878.) London: J. and A. Churchill.

chamber arise? The microscope shows that it is caused by the pressure of the swollen ciliary processes against the posterior surface of the iris.

e. Can we, then, accept Weber's explanation of glaucoma, viz., that it is the result simply of swelling of the ciliary processes? This explanation, although certainly a nearer approach to the truth than any before given, is incomplete. In the first place, what causes the swelling of the ciliary processes? Not, as Weber asserts, the presence of those maladies,—cardiac, vascular, and pulmonary—which induce general venous turgescence, for these diseases even in their severest forms are but rarely associated with glaucoma, and in many cases of glaucoma they are entirely absent. In the second place, a simple swelling of the ciliary processes would, by reason of the relation of the parts concerned, tend to displace the lens backwards and to deepen the anterior chamber, whereas in glaucoma the tendency is in the opposite direction.

f. Can a closure of the angle of the anterior chamber be brought about by pressure upon the ciliary processes from behind, in the absence of swelling in the processes themselves? A series of experiments upon the fresh dead eye of the pig and the human subject established the fact that a very slight excess of pressure in the vitreous chamber suffices to drive the ciliary processes forward against the periphery of the iris, and to close the angle of the anterior chamber, the closure being so complete that fluid in the anterior chamber, even when under a pressure much greater than that proper to the eye, can find no exit. This result presented a close simulation of the conditions which exist in glaucoma, viz., a slight advance of the lens in association with a complete closure of the angle of the anterior chamber. It appeared to indicate that the primary cause of glaucoma is some condition which induces a slight excess of vitreous pressure over aqueous pressure.

g. A variety of considerations, too lengthy to be repeated here, led to the conclusion that a current of intraocular fluid

is constantly passing from the vitreous chamber to the aqueous chamber, by osmosis through the suspensory ligament, where it bridges across the interval between the ciliary processes and the margin of the lens,—the "circumlental space." Any obstruction in the path of this current would, it appeared certain, cause an excess of pressure in the vitreous chamber, and induce a condition of things similar to that which exists in glaucoma.

h. Narrowing of the circumlental space suggested itself as a possible cause of excess of pressure in the vitreous chamber. The following considerations, amongst others, favoured the idea that this is actually the cause of glaucoma:—Glaucoma is pre-eminently a disease of advanced life. In advanced life the lens increases in diameter, and thereby presumably narrows the circumlental space. Injuries of the lens, which cause it to swell as a whole, frequently induce glaucoma. Swelling of the ciliary processes is undoubtedly a part of the glaucomatous process. This swelling must tend to narrow still further the circumlental space.

A definite theory as to the origin of primary glaucoma was therefore formulated in the following words:—

The cause of primary glaucoma is a diminution of the distance which separates the margin of the lens from the ciliary processes. Two factors combine to produce this diminution, viz.:—

1. *Increase of the diameter of the lens.*
2. *Swelling of the ciliary processes.*

The former is usually, if not invariably, the primary change, and is itself the indirect cause of the latter.

For a full statement of the evidence on which this theory was built up, I must refer to the chapters on pathology in my previous essay. There also will be found a discussion of the validity of the theory in relation to various points in the etiology of the disease, to the chronic and acute types, and to the curative action of iridectomy. Some facts also which are at first sight irreconcileable with the obstruction theory

of glaucoma, but which in reality tend towards its corroboration, are there considered.

The object of the present communication is to record certain recent observations which bear upon the several points in question.

Further evidence as to the escape of the intraocular fluid at the angle of the anterior chamber is, perhaps, hardly necessary, since Leber's discovery is now generally accepted. It has recently received confirmation in the experiments of Schoeler.* He experimented upon the dead human subject, and upon rabbits under chloroform and after death. Eschars were produced upon the surface of the globe by the actual cautery, and the internal pressures and the rates at which fluid escaped were measured by the manometer. No retardation of the escape of fluid occurred except when the ciliary region was the part cauterised. In the latter case a very marked retardation was invariably observed. Neither occlusion of the vortex veins nor cauterisation of the optic nerve entrance produced any such retardation. (The manometrical measurements invariably indicated an equality of pressure in the aqueous and vitreous chambers.) Again, Leopold Weiss,† by means of injections into the vitreous chamber in living animals, and the subsequent treatment of the enucleated eye with chemical reagents, has traced the transit of the injected fluid to the angle of the anterior chamber, and thence to the exterior of the globe. In a recent case of my own, high tension was set up in a blind eye by a blow. After excision, I found the angle of the anterior chamber uncompressed, but blockaded throughout the whole of the circle by aggregated blood corpuscles, the result of hæmorrhage into the chamber.

The assertion that a current of intraocular fluid is constantly passing from the vitreous to the aqueous chamber, through the circumlental space, was based, so far as my own

* Graefe's Archiv., xxv, iv. See also Lond. Medical Record, March 15, 1880, p. 114.

† Graefe's Archiv., xxv, ii,

observations went, chiefly upon evidence of a negative character. Since that time Deutschmann has pointed out* that if the aqueous chamber of the human eye be evacuated by puncture shortly after death, it is refilled within a few hours. Seeing that the same thing occurs in the freshly excised eye, the only possible source of the regenerated fluid is the vitreous humour. I have lately performed this experiment myself several times, and always with the result stated. Again, the experiments of Weiss, already referred to, indicate a current in this direction.

It will not now be denied by any experienced observer that in eyes blinded by primary glaucoma some compression of the angle of the anterior chamber is constantly discoverable with the microscope. The solid adhesion of the two surfaces which exists in the great majority of specimens is, however, occasionally absent. Of five additional specimens lately examined by myself, an adhesion is present in four. In the fifth, in which the angle of the chamber appears, under a low power, to be in a normal condition, a higher power proves beyond a doubt that the surfaces have previously been in contact, if not adherent. Cells and pigment from the surface of the iris adhere to the cornea throughout precisely that region which in most cases is the site of the adhesion. Drawing 11 exhibits a section taken from this specimen. The eye unfortunately lay for many weeks in Müller's fluid before it was opened for examination, under which treatment shrinking of the lens invariably occurs. I think that the separation of the periphery of the iris from the cornea which has occurred in this case was a *post-mortem* change produced by shrinking of the lens and traction of its suspensory ligament upon the ciliary processes. The wide separation of the iris and cornea occurred in part during the process of mounting, which in sections of this kind is unusually difficult, by reason of the slender attachments of the lens. It is worthy of notice that in this eye only of the five in question the pupil remained con-

* Graefe's Archiv., xxv, i.

sensually active for many months after blindness was complete, in fact until a short time before excision. (The case was one of very slow, painless, non-irritative glaucoma, of perhaps ten years' duration before the hyperæmia and pain occurred which called for excision.)* This suggests the inference that so long as the iris retains its consensual activity, and so long perhaps as it reacts freely to myotics, the angle of the chamber is not compressed so forcibly as to produce a firm adhesion, an inference which accords with the fact that it is chiefly during this stage of the malady that iridectomy is able to effect a permanent reduction of tension.

Again, I think it will not be disputed that the closure of the angle of the anterior chamber is directly due to the pressure of the ciliary processes against the iris, as originally asserted by Adolph Weber.† In some cases, truly, the processes are found small, retracted, and far removed from any contact with the posterior surface of the iris, but there is ample evidence to show that they have previously occupied a much more advanced position; *e.g.*, the relations of the parts in the healthy eye (Drawings 1 to 5); the invariable limitation of the adhesion to a region corresponding with these relations; my own experiments on this point;‡ the close and forcible contact, even the adhesion, of the processes with the iris discoverable in the more recent cases of glaucoma; the fact that in certain of the specimens in which the processes are greatly retracted the iris retains the impress of their previous pressure, and that detached portions of the processes remain adherent to the iris in such cases. The glaucoma sections in my own possession present the greatest differences in the size and position of the ciliary processes, from extreme turgescence on the one hand, to extreme retraction on the other: I believe, nevertheless, that the closure of the angle of the anterior chamber has been produced in every

* "Glaucoma: its Causes, &c., &c." Case 21, p. 256.

† Graefe's Archiv., xxii, i.

‡ "Glaucoma: its Causes, &c.," p. 127.

single instance by the pressure of the processes against the iris. I speak here of secondary as well as of primary glaucoma. This matter will come again under notice in a subsequent paragraph.

I come now to the examination of certain points essentially involved in my own theory. The questions on which it appeared necessary to obtain further information were these:—

1. Is it a fact that the diameter of the lens increases with age?

2. Does an increase in the diameter of the lens necessarily diminish the circumlental space?

3. Is the circumlental space actually narrower in the glaucomatous eye than in the healthy eye?

4. If so, is the narrowness due to an abnormality in the lens or in the ciliary processes, or in both?

I shall attempt to give a categorical answer to each of these questions, and to substantiate it with evidence.

1. *The diameter of the lens increases with age.*

It is easy to convince oneself of this fact by examining with the ophthalmoscope a series of cases in which iridectomy has been performed. In the eye of the child and in that of the young adult the margin of the lens is always readily visible through the gap in the iris, while in the senile eye, if discoverable at all, it is only to be seen by a much more oblique inspection, and is manifestly much more nearly under cover of the sclera than in the former case. In eyes which have been iridectomised for glaucoma the margin of the lens is almost always, in my experience, entirely removed from view. In citing this comparison I do not overlook the fact that an advance of the lens, such as generally occurs in glaucoma, tends of itself to make the margin less accessible to ophthalmoscopic inspection, or that the existence of an arcus senilis renders an eye unsuited for examination in this respect.

In order to obtain more exact information as to the size

of the lens and its relation to the ciliary processes at different periods of life, I examined ten healthy eyes (five pairs) removed shortly after death, and prepared in such a way as to reduce to a minimum any shrinking or disturbance of the parts in question.

The following was the mode of examination:—

The specimen, after lying for seven days unopened in Müller's fluid, was divided by a circular incision, corresponding roughly with the ora serrata. The anterior portion, containing the lens and ciliary processes intact, was placed in solution of hydrate of chloral 1 in 20. The remnants of the vitreous humour and hyaloid membrane were carefully removed. The diameter of the lens was measured in several meridians by means of finely-pointed spring compasses and a magnifying glass, as the specimen lay in the fluid. Some days later, during which time the lens underwent no perceptible change of size, the specimen was placed in a small covered china dish, containing just enough thick gum-water to cover it, and at once frozen solid by immersion of the vessel in a mixture of ice and salt, and then divided into halves by a meridional section, and returned to the chloral solution. The thickness of the lens was then measured with compasses. Placed with the cut surface looking upwards, the specimen was readily examined with a low power of the microscope as it lay in the chloral solution. By means of a special arrangement of the microscopic camera, and a vertical stage carrying a sheet of paper upon which the image of the object was seen projected, I made an accurate drawing, or rather tracing, of the cut surface of each specimen enlarged exactly ten diameters. The series of sepia drawings thus obtained have been reproduced, in half size, by photography, and printed in *fac simile* by the Woodbury Photographic Printing Company of 157, Great Portland Street, London. A millimetre scale was added to each of the original drawings to facilitate measurement, and to serve as a test of the accuracy of the photographic reduction. Finally, the specimens from which the drawings were made were mounted in glycerine jelly, with special precautions against shrinking, and are now preserved. Repeated examinations of the specimens at different times and under slight variations of the method of treatment lead me to believe

that a slight contraction of the lens occurs in spite of all that can be done to prevent it.

Drawings 1, 2, 3, 4, 5, exhibit the parts in question in a series of healthy eyes prepared as above described. The ages of the subjects were respectively 21, 26, 49, 65, and 90. It is manifest at first glance that in this series the size of the lens increases with age. The following table shows the actual measurements of each lens as obtained by means of compasses and a magnifier:—

Number.	Age.	Diam. of lens.	Thickness of lens.
1	21	8·0 mm.	5·5 mm.
2	26	8·5 ,,	5·5 ,,
3	49	8·75 ,,	5·75 ,,
4	65	9·75 ,,	6·25 ,,
5	90	9·75 ,,	6·5 ,,

These measurements will be found to agree very closely with those afforded by the drawings; where any discrepancy exists it must be attributed, I think, to the difficulty of ensuring an absolutely identical focus of the microscope in every instance, failing which minute variations in the degree of enlargement are inevitable. The fellow eyes of those here represented were examined also, and gave almost absolutely identical measurements in every instance. I also measured a lens which escaped intact in its capsule from an eye with necrosed cornea during the operation of excision. The patient was a man aged 29; the size of the lens corresponded closely with that in No. 2 of the above series. The difficulty of obtaining specimens has hitherto prevented me from extending this series of observations, but I think that even the small number here given, taken in conjunction with the ophthalmoscopic observations previously mentioned, afford sufficient evidence that the diameter of the lens increases with age. It would appear, further, that this increase in diameter is associated not with a general flattening and a loss of thickness, as I had previously supposed, but with an

increase of thickness: in short, that the lens undergoes a general increase of bulk with the advance of life.

2. *As the diameter of the lens increases the distance which separates it from the ciliary processes decreases: the circumlental space diminishes with age.*

It would appear possible, *à priori*, that an enlargement of the lens might, by relaxing the suspensory ligament, lead to a retraction of the ciliary processes, and that thus the circumlental space might remain unchanged in width. The drawings show that such a retraction does not occur. The close proximity of the edge of the lens, and the ciliary processes in Drawings 4 and 5, contrasts strongly with their separation in 1 and 2. From the appearances visible with the microscope, in glaucomatous eyes especially, it is evident that the apices of the processes are, to a certain extent, free to advance when turgescent, and to retract when empty, independently of the traction exercised by the suspensory ligament; and, further, by placing a specimen in glycerine and water, it is easy to prove, in the case of the dead eye at least, that contraction of the lens widens the circumlental space without any rupture of the suspensory ligament. It is probable that in every case the lens assumes a slightly more globular form after death than it has during life, and we should expect this alteration to be greater in the youthful than in the senile lens; in fact, we probably have evidence of its occurrence in those specimens in which the ciliary muscle is partly detached from the sclera by the traction of the suspensory ligament; but, having regard to the contour of the lenses in my series, it does not appear that the variation in the circumlental space can be due in any great degree to a *post-mortem* change of form in the lens.

3. *In glaucomatous eyes, after excision, the circumlental space is sometimes widely open: but there is usually evidence to show that it has previously been abnormally narrow.*

The first glaucomatous eye which I examined with regard to this point appeared, at first sight, to disprove the theory

at stake. It is represented in Drawing 7, and one side of the other half of the specimen is shown on a larger scale in Drawing 8. The circumlental space, instead of being narrowed or abolished, is here more widely open than in the healthy senile eye. It has been shown in a previous paragraph that the closure of the angle of the anterior chamber which is found in these cases is produced by the pressure of the ciliary processes against the iris. In this specimen the ciliary processes are far removed from the posterior surface of the iris, and do not reach nearly far enough forwards and inwards to account for the adhesion which here exists. The iris, nevertheless, bears unmistakeable traces of having been compressed by the processes at some previous time. In its uveal surface is a very significant concavity (see Drawing 8), limited internally by a protuberance of the uvea and by a general thickening of the whole membrane, which mark the point at which the compression ceased. The solid adhesion of the iris to the cornea is just coextensive with the compressed portion. (These details are seen with convincing clearness in the specimen itself.) Further, it must be noted that the distance between the apices of the opposite processes (Drawing 7) is greater by about 2 mm. than in any one of the healthy series; and also that the body of the ciliary muscle slopes away from the lens in a way which contrasts strongly with its prominence in every one of the healthy eyes. Now if we imagine the atrophied ciliary muscle in this drawing to be replaced by one of normal size and shape, and the retracted ciliary processes to resume a normal prominence and position, taking in each case the healthy eye as a guide, it is evident that a very much narrower circumlental space would remain; and if we go a step further and imagine the processes to be swollen, to be applied to the compressed portion of the iris, and to extend somewhat beyond the limit of the adhesion (see Drawings 6 and 12), and the lens to be pushed somewhat forward, so as to touch the iris, which it always does during life, then, certainly, the circumlental space

would be nearly, if not entirely, abolished. These supposed changes may be advantageously studied by means of a piece of tracing paper laid over the drawing.

How are we to explain the extreme retraction of the ciliary processes which is present in this and similar cases? Is it the result of the gradual suppression of their secreting function,* and of the long continued pressure upon their external surface, or is it a *post-mortem* change? Some retraction of the processes occurs probably at the moment of excision. The operation suddenly cuts off the arterial supply to the globe, and at the same time promotes the escape of blood from its interior by the division of a host of veins upon the surface, and this at a moment when the intraocular pressure is still at a high pitch; a rapid and forcible emptying of the internal vessels is the consequence. This is made manifest by the excessive injection of all the superficial vessels which occurs in glaucomatous eyes immediately after excision. This cause of retraction of the processes finds no parallel in the conditions which are present in the healthy eye at the moment of death. It occurred to me that if this explanation were the true one, the retraction might be avoided by puncturing the sclera, and thereby reducing the intraocular pressure prior to excising the eye. I carried out this idea in the next case which occurred in my practice. The specimen is shown in Drawing 6. The processes are large and prominent, and under the microscope all the internal vessels of the eye, those of the processes included, prove to be fully injected with blood. The large size of the processes appears, nevertheless, to be due rather to proliferation of the perivascular connective tissue than to distension of the vessels themselves. The difference between the processes in Drawings 6 and 7 cannot, therefore, be attributed in any great degree to the puncture in the sclera, and the consequent injection of the vessels; it has more connexion, probably, with the fact that the glaucoma was in the one case acute in character, in

* "Glaucoma: its Causes, &c.," p. 163.

the other non-irritative and chronic. A repetition of the foregoing experiment in one or two cases of the latter class would decide the question.

In these two specimens (Drawings 6 and 7) the lens now lies at some distance from the iris, but the contour of the latter shows that the lens has previously held a more advanced position in contact with it. If we imagine the lens in Drawing 6 pushed forwards against the iris, the circumlental space disappears; and supposing the processes of the right side to be advanced sufficiently to account for the adhesion at the angle of the anterior chamber, their close compression between the lens and iris is inevitable.

Drawing 12 exhibits a section from Specimen 6, in which a part of the apex of the process is thrust forward in a wedge-like form very suggestive of compression between the lens and iris. It protrudes, be it observed, not merely as far as the limit of the adhesion of the iris to the cornea, but considerably beyond it. It appeared probable that the existence of such a compression would involve some lateral swelling of the processes—some diminution of the space which separates each process from its neighbour. To test this point, I made a series of sections transversely through the processes at and near to the angle of the anterior chamber. Drawing 13 represents such a section from the healthy eye of the subject 65 years of age (No. 4). It was taken just within the angle of the chamber, so as to include a portion of the periphery of the iris, and passed very obliquely, as the drawing shows, through the ligamentum pectinatum and Schlemm's canal. In the centre are three processes cut through transversely just at the point where their uveal covering is continuous with that of the iris. Sections anterior to this point cannot be mounted, for the heads of the processes being severed from their attachments drop out; those at each side of the drawing are of course seen in oblique section. Drawing 14 shows a section from a similar or a slightly less peripheral situation in a glaucomatous eye, the specimen previously referred to (Drawing 11) in which the angle of

the chamber, though open, shows traces of previous closure; the patient's age was 70. The large size and the tortuous mutually-moulded outlines in the latter case present a significant contrast with the smaller size, the simpler contour, and the wider interspaces of the healthy processes. The tortuosity would probably have been still more extensive had the sections been made in a direction perpendicular to the surface of the globe, instead of parallel with its antero-posterior axis; they would then have passed through that part of each process which presented directly towards the lens, whereas they now pass directly backwards through that portion which lies in the groove of the suspensory ligament, and thus do not fairly represent the condition of the circumlental space. The close mutual compression which has existed at the upper part of these processes would manifestly aid in the complete obliteration of the circumlental space.

Again, in Drawing 11, taken from this same specimen, the appearances indicate with considerable show of probability that the edge of the lens has previously been in contact with the processes, its subsequent retraction having been caused by the prolonged action of the preservative solution, as explained in a former paragraph. This probability rises almost to a certainty when the section is examined under a high power. The margin of the lens between the two points of attachment of the suspensory ligament is thickly studded with cells or cell-like bodies, similar to those which coat the surface of the process.

Drawing 9 has not yet been referred to. It represents a specimen of chronic glaucoma. The course of the disease had been extremely slow. The eye had been blind for four years previous to excision; neither pain nor redness had occurred until the last three months. The onset of irritative symptoms called for excision. In the fellow eye chronic glaucoma had been permanently arrested by iridectomy. On the right side of the drawing the extent of the closure of the angle of the anterior chamber indicates a previous position of the processes which would have closed the circumlental

space. On the left side the processes are further removed from the lens, and the closure of the angle of the chamber is of much smaller extent. These relative differences are interesting as indicating the import of the proximity of the margin of the lens. In view of the slow progress and non-irritative character of the malady in this case, and in the absence of evidence that the lens has been driven forwards, it seems not unreasonable to conclude, in accordance with the appearances shown in the drawing, that the interference with the circumlental space has been less complete or less universal than in the two former cases.

Drawing 10 represents a specimen of glaucoma, the history of which was altogether exceptional. The patient, a woman, æt. 63, stated that four years ago her sight was good, when one day, while carrying a bucket of water, she felt a sudden sharp pain in the left eye, which lasted only a few minutes. Blindness was immediate and permanent. Pain occurred from time to time ever after, and set in severely three weeks ago. The other eye presented an advanced stage of chronic glaucoma, with recent irritative symptoms. The blind eye, hard, red, and painful, was excised at once; iridectomy was performed upon the other with good result. The specimen when opened showed a deeply cupped disc, proving the excess of tension to have been of long duration. The suspensory ligament, to the naked eye, was thickly coated with what appeared to be a greyish semi-opaque deposit. This proved under the microscope to be the remnant of an old hæmorrhage. Large quantities of blood corpuscles, held together apparently by a fibrinous coagulum, coated the whole of the suspensory ligament and adjacent processes; a fibrinous film containing blood corpuscles lined the cornea loosely; and corpuscles were aggregated in the neighbourhood of the angle of the anterior chamber. No trace of their natural colour remained. The ostensible starting point of the glaucoma in this case was doubtless a hæmorrhage from some part of the ciliary processes, causing sudden loss of sight; but seeing that the fellow

eye was subsequently attacked by chronic glaucoma, it is not impossible that conditions predisposing to glaucoma, or even the first stage of the malady itself, may have preceded the hæmorrhage. Whether the circumlental space was ever abnormally narrow in this eye (as indeed it now appears to be in some parts of the circle), or whether it was obstructed by the blood which everywhere thickly coats the suspensory ligament, and chronic glaucoma induced thereby, are questions for conjecture only. The specimen affords little evidence as to the point under discussion, but inasmuch as the condition of the fellow eye leads me to class it as one of primary glaucoma complicated with hæmorrhage, it cannot fairly be omitted from the series.

Adolph Weber, in his invaluable work on the cause of glaucoma, already referred to, states that it is not unusual after iridectomy to find a ciliary process apex or a pigment mass adherent to the equator of the lens, as evidence of the previous swelling of the processes. I have not yet met with a well marked example of this, but I have more than once seen peculiar changes in the transparency of the capsule at the equator, or rather just in front of the equator, which appeared likely to have been caused by the previous contact and pressure of the processes.

In spite of the difficulty of ascertaining the precise relation which the lens holds to the ciliary processes in the living eye, I think that the evidence here set forth goes far towards proving that in certain stages of glaucoma the circumlental space is unduly narrow.

4. *There is at present no sufficient evidence to show whether the abnormality of the glaucomatous eye is primarily in the lens or in the ciliary processes.*

If the substance of the foregoing paragraphs be true, it may be safely asserted that the comparatively large diameter of the senile lens is an essential factor in the production of glaucoma, or in a certain sense, that an increase in the size of the lens is the primary cause of the disease. But inasmuch as this increase is common to all senile eyes, whereas

glaucoma is an exceptional condition, we have yet to ascertain the additional factor which determines its occurrence. Excluding the case in which the circumlental space appears to have been occluded by hæmorrhage, I have as yet measured the diameter of the lens in only three specimens of primary glaucoma (Drawings 6, 7, and 9). In each of these, and especially in the two first, the lens has a diameter distinctly greater than that of the largest in the healthy series; and it is especially noteworthy that neither of these two glaucomatous lenses would lie between the ciliary processes in any one of the healthy series. This may be proved by measurement of the drawings with a pair of compasses. Should further observation show that the lens of the glaucomatous eye has, on the average, a greater diameter than that of the healthy eye at the same period of life, one starting point of glaucoma would, I think, be finally demonstrated. Meanwhile the isolated measurements afforded by my specimens will justify no general conclusion. It is not probable that the lens is of excessive size in every case. When the circumlental space in the healthy eye is reduced to such narrow dimensions as those shown in Drawing 5, a slight enlargement of the ciliary processes would suffice to establish the "vicious circle" of the glaucomatous process. Hence, I am disposed to restate the inference suggested in my previous essay, viz., that an undue size of the lens is ordinarily the predominating factor in the chronic form of glaucoma, vascular engorgement in the acute. The conditions under which such engorgement may arise, and the mode in which an excess of tension tends in any case to perpetuate and aggravate itself by inducing passive hyperæmia in the ciliary processes (Weber) were fully considered in the same work.

A critic has objected to my statement that a shallow anterior chamber is a constant feature of primary glaucoma. The objection is just, for the rule is undoubtedly not without its exceptions; and on the strength of these exceptions it must be admitted that an excess of vitreous pressure over aqueous pressure is not invariably present.

It is probable that in certain cases the lens may touch the ciliary processes and compress the angle of the anterior chamber without arresting the flow of fluid from the vitreous to the aqueous chamber; as, for example, in lateral luxations of the lens, in which manifestly the obliteration of the circumlental space at the one side involves its greater patency at the other. In like manner a slightly excentric position of the senile lens (see Drawing 9) may, perhaps, occasionally be the cause of glaucoma. In such cases no advance of the lens would occur, because a portion of the circumlental space would remain patent, and an excess of vitreous pressure over aqueous pressure would not arise. So far, then, as these exceptional cases of normal or deep anterior chamber are concerned, my description of the glaucomatous process needs modifying. A shallow anterior chamber is, however, the rule; hence a general explanation of the disease must include not merely a contact of the lens with the ciliary processes, but an obstruction to the flow of fluid through the circumlental space.

No reference has been made in the present paper to the recent researches of Brailey in the pathology of increased tension.* His discoveries are both important and interesting, and, so far as my observation has gone, find confirmation in my own specimens; but inasmuch as the changes in the blood-vessels and ciliary muscle to which he has drawn attention are, in my opinion, consequences rather than causes of increased tension, they find no place in the present inquiry. To the original and most instructive work of Adolph Weber† I desire again to express my obligation.

In conclusion, it will not be out of place again briefly to summarise the views which I have put forward in the present and in the previous essay, concerning the origin of Primary Glaucoma.

The fundamental and essential cause of glaucoma is an

* R.L.O. Hosp. Reports, vol. ix, parts 2 and 3.

† Graefe's Archiv., xxii, i.

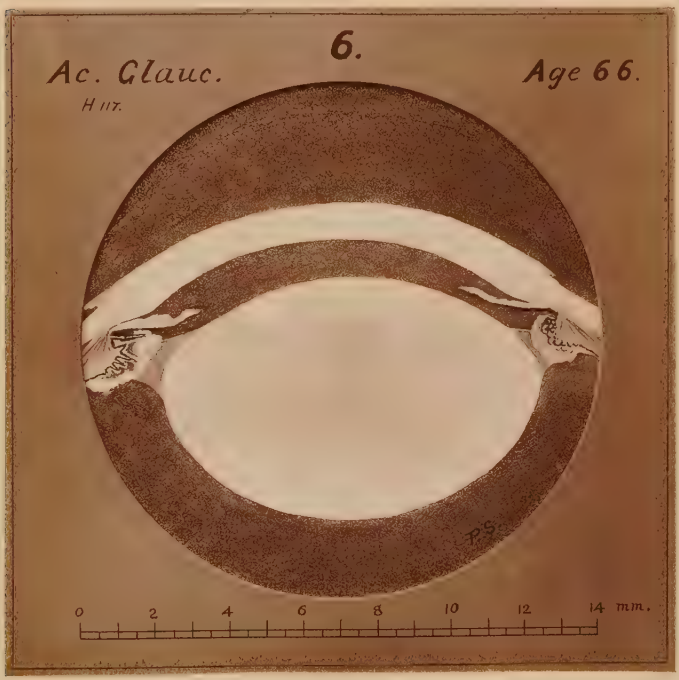
Healthy
1.
Age 21
P.S.
0 2 4 6 8 10 12 14 mm.

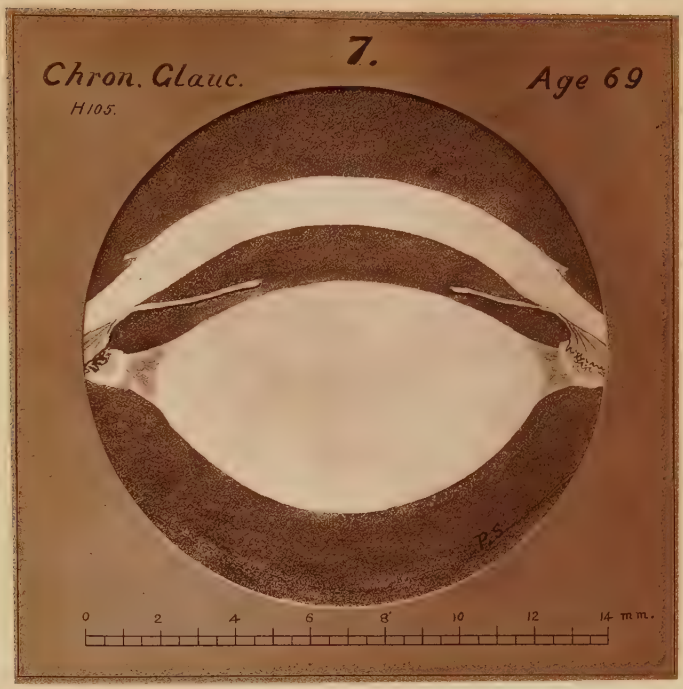
Healthy
2.
Age 26
P.S.
0 2 4 6 8 10 12 14 mm.

Healthy
3.
Age 49
P.S.
0 2 4 6 8 10 12 14 mm.

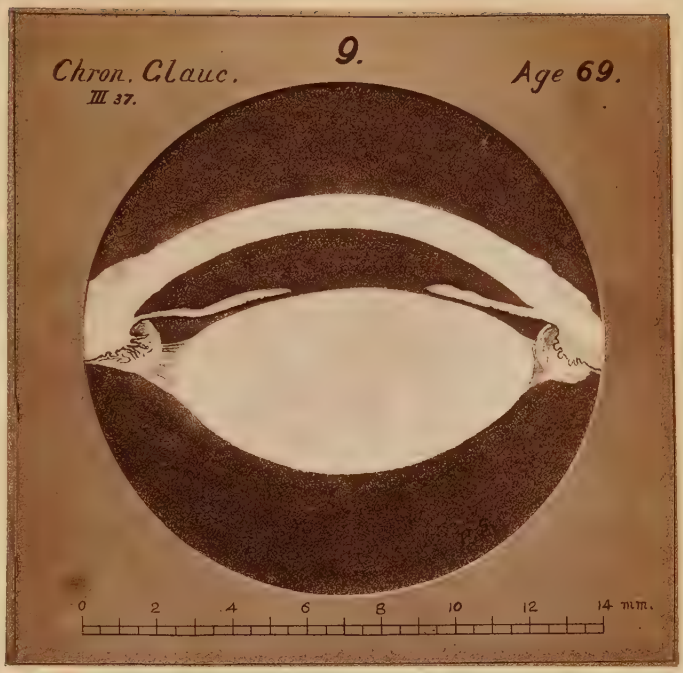
Healthy
4.
Age 65
P.S.
0 2 4 6 8 10 12 14 mm.

5.
Healthy
Age 90
P.S.
0 2 4 6 8 10 12 14 mm.


6.
Ac. Glauc.
H 117.
Age 66.
P.S.
0 2 4 6 8 10 12 14 mm.


7.
Chron. Glauc.
H 105.
Age 69
P.S.
0 2 4 6 8 10 12 14 mm.

8.
Chron. Glauc.
H 105.
Age 69.
P.S.
0 1 2 3 4 5 6 7 mm.


9.
Chron. Glauc.
III 37.
Age 69.
0 2 4 6 8 10 12 14 mm.

10.
Glaucoma.
H 283.
Age 63.
0 2 4 6 8 10 12 14 mm.

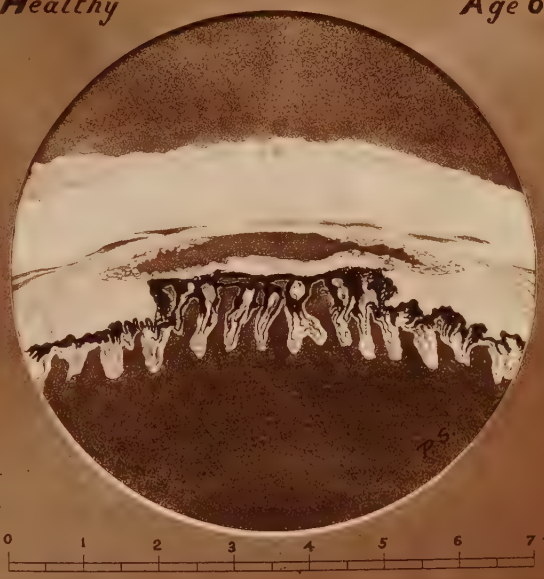
11.
Chron. Glauc.
F 174.
Age 70.
P.S.
0 1 2 3 4 5 6 7 mm.

12.
Ac. Glauc.
H 117.
Age 66.
P.S.
0 1 2 3 4 5 6 7 mm.

13.

Healthy

Age 65.

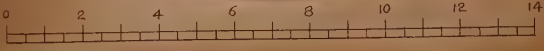


14.

Chron. Glauc.

F. 174.

Age 70.



abnormality situated in the eye itself, namely, an insufficiency of space between the ciliary processes and the lens.

All conditions which tend to promote venous turgescence, arterial hyperæmia, or increased secretion within the eye, may become exciting causes of glaucoma, provided that this abnormality is present.

ON CENTRAL AMBLYOPIA.

By George W. Berry, M.B.

That form of amblyopia in which direct vision is impaired alone, whilst that of the peripheral portions of the retinæ is entirely unaffected or but slightly less acute than normal, constitutes a group of cases which claim consideration as well from their relative frequency as from the fact that the prognosis is all but invariably good. They very rarely end in complete blindness, occasionally they remain stationary after more or less loss of direct vision, and, in the great majority of instances, they undergo partial or complete recovery. Their diagnosis is, therefore, a matter of importance, and, I cannot but think that were they always recognised as a distinct class, we should hear less of the wonderful cures produced by strychnia and electricity in amaurosis or optic nerve atrophy.

Cases of central amblyopia present themselves for treatment at Moorfields almost daily. Through the courtesy of the surgeons I have had frequent opportunity of studying them, besides having had my attention drawn to the subject at the clinic of Dr. Edmund Hansen, at Copenhagen, where they have been carefully studied for the last thirteen years, and from whence 183 cases constituted the material for an admirable treatise by Krenchel* in 1876. I would, therefore, as briefly as possible, point out what appear to me to be the chief points of importance in the diagnosis and prognosis of this form of amblyopia without giving any cases in detail. Illustrative cases will be found in Mr. Hutchinson's last paper on Tobacco Amaurosis,† and Mr. Nettleship‡ has given very accurate representations of the field of vision with reference to the central colour scotoma to which, so far as

* "Amblyopia Centralis." Copenhagen, 1876. (Abstract in Nagel's Jahresbericht for 1876.)

† Ophthalmic Hospital Reports, vol. viii, p. 486.

‡ St. Thomas's Hospital Reports, 1879.

I am aware, he was the first to draw attention in this country.

The existence of cases in which the defect of vision was owing to the presence of a central scotoma has been long known, and constitutes one of the classes of cases described by v. Graefe* in his famous lectures on amblyopia, but these were the more severe cases only in which central vision was not only relatively but absolutely worse than the peripheral. It was not until Leber† and Förster‡ discovered the means of detecting slight central defects by examining the colour sense that many cases that had come under some such category as that of amblyopia *sine causâ* were really found to belong to the central group of cases where the amblyopia was probably of toxic origin, and in which no central colour scotoma could be detected.

The first of Graefe's§ illustrative cases was evidently one of central scotoma, and probably due to tobacco poisoning, it is described as a case of the "curable form of congestive amblyopia with normal field of vision."

Central amblyopia appears in the great majority of instances to be a toxic affection most commonly caused by the abuse of tobacco. The occasional deleterious effect of tobacco on vision has long been known or rather suspected; it is referred to by Mackenzie in his well-known treatise on the diseases of the eye, and later on in 1863 by Wordsworth,‖ Hart,¶ and Hutchinson.** At that time, however, no systematic examination of the field of vision appears to have been generally practised, and consequently no thorough classification of such cases into a distinct group was possible.

The general features of these cases of central amblyopia are probably familiar to most ophthalmic surgeons. A man,

* *Vide* Zehender Klin. Monatsbl., 1865.
† Graefe's Archiv., vol. xv, part ii, p. 26, 1869.
‡ Ein Jahresbericht der Schls. Ges., 1868. Heidelberg Congress, 1871.
§ *Loc. cit.*, p. 193.
‖ Medical Times and Gazette, April 4th, 1863.
¶ Lancet, July, 1863.
** *Ibid.*, November, 1863.

generally beyond thirty years of age, complains of gradual failure in the sight of both eyes, accompanied by a disagreeable sensation of fog; usually this has been going on for some months before he applies for advice; he has seen well four or six months ago, but latterly he has not been able to read even large print, and has finally been obliged to give up his occupation. He is often alcoholic, not unfrequently has lost his appetite, or is sleepless, has suffered from diarrhœa, hæmorrhage from piles or some other drain on the system, but often he has noticed nothing unusual in his state of health; very frequently indeed he is a great smoker. There are still some ophthalmologists* who do not appear to be convinced of the toxic effect of tobacco alone in producing amblyopia, and ascribe the defect of vision more to alcohol, to which they assert patients with so-called tobacco amblyopia are always addicted. But why then should improvement take place so rapidly and so surely in many cases when the supply of tobacco consumed is diminished or altogether stopped? Again, although it must be admitted that it is very rare indeed to find one of these favourable cases of central amblyopia in an individual who smokes immoderately without at the same time being in the habit of occasionally drinking to excess, yet I have seen two typical cases, one an old man aged 70, who had been a tee-totaller for forty years, and the other a boy of 19 (the youngest patient in whom I have met this form of amblyopia), who certainly did not drink; and similar cases have been met with by others. Some cases, however, appear to be complicated with chronic alcoholic poisoning, and I think as a general rule it will be found that the more complete the alcoholism, the slower and more imperfect is the recovery on the cessation of smoking. Mr. Hutchinson is of opinion that the effect of alcohol is, on the whole, antagonistic to tobacco, and in this I am inclined to think he is right, judging from personal experience and from the experience of others whom I have questioned on the subject, and who, like myself, are susceptible to the depressing

* *Vide* Lawson's "Manual of Diseases of the Eye."

effect of tobacco in any quantity or of more than moderate strength. But we must draw the distinction between alcoholic stimulation and alcoholism, where the vital powers are actually lowered and notoriously unable to withstand any unusual strain. It is just possible then that the toxic effect of tobacco may not assert itself until the antagonistic effect of the alcohol has from over stimulation given place to a certain amount of depression and the equilibrium thus destroyed. This would appear sometimes to explain why a man who has smoked all his life with impunity is all at once affected by tobacco. Although it by no means explains the cause of central amblyopia in all cases, as on the one hand we meet with cases, already referred to, where no alcohol is used, and on the other hand a few cases occur in which the alcohol itself must be looked upon as having a more direct effect on the production of the symptoms. Anyone with any experience of smokers must have been struck by the different degrees in which different men are affected by tobacco, how some never smoke anything but mild tobacco, and seldom, except after meals, whereas others smoke the strongest tobacco at any time of the day. The fact of such idiosyncrasy being a matter of daily experience, is strongly suggestive of most cases of tobacco amblyopia being found amongst the former class. The class of patients who apply at the hospital (and it is in this class that central amblyopia is most frequent), generally smoke shag, and are often obliged to do so on an empty stomach, and although occasionally they confess to have great difficulty in learning to smoke, and being somewhat susceptible to tobacco altogether, still this cannot always be elicited.

The evidences then in favour of tobacco being the agent apart from alcohol in the production of the majority of cases of central amblyopia, are the gradual disappearance of the symptoms on the cessation of smoking without any other treatment being resorted to, and frequently without the supply of alcohol being diminished, and the occasional presence of exactly the same symptoms in individuals who do not use

alcohol at all. To these may be added the very suggestive fact of its extreme rarity in women, except in cases where there is distinct palor of the optic disc, and where the subsequent course confirms the diagnosis of progressive atrophy.

The subjects of central amblyopia often seem to suspect that their ailment is due to smoking, and express great willingness to give it up. Frequently, however, their good intentions are left unexecuted.

They invariably complain of great mistiness of vision, which varies considerably at different times, and is almost always said to be less in a dull light ; they see " through a fog ;" this is entirely confined to the region of the central defect, and we are frequently told that things appear much clearer when seen eccentrically. The improvement in vision in a dull light is probably only relative, that is, the difference between the indistinctness of direct vision and the clearness of indirect vision is not so marked, besides which the pupils are perhaps a little more dilated, although they are often not small even in a bright light. I have at any rate not been able to demonstrate any actual increase in visual acuity in subdued light ; on the contrary, I have always found the reverse, and in one case which I examined with Masson's disc, by which means we are able to test the power of distinguishing between shades of grey differing only slightly in intensity, the light-sense was decidedly best when the absolute intensity was considerable (in bright daylight), and diminished in proportion as it decreased. This was tried with and without a stenopaic aperture (2 mm. in diameter). My observations in connexion with the light-sense in these cases are, however, too few to enable me as yet to draw any general conclusions, and, indeed, the examination of the light-sense of a definite portion of the retina is a matter of no inconsiderable difficulty. Some interesting experiments on this subject will be found in Krenchel's paper above referred to.

Usually the visual acuity has become very materially diminished before these patients apply for advice (Jæger, 14

to 20 at 8″), but we meet with all degrees, and it is just in the minor degrees, especially if there be at the same time some complication (as for instance incipient cataract or central choroido-retinitis), that there is any difficulty at all in the diagnosis. The amblyopia is generally very much the same in both eyes; when there is any considerable difference, I have, as a rule, found it to be in the higher degrees. If the acuity be as low as J. 19 or 20, it will often be found that the eccentric vision is better than the direct, and it is important to bear this in mind, otherwise an improvement may often be assumed when, in reality, the patient has only discovered how to supply the defect of his central vision by making use of the peripheral portions of the retina. Besides, according to Graefe,* when the direct vision has become very defective, an improvement in acuity amounting to double or even treble the original amount often takes place in that of the peripheral portions of the retina immediately surrounding the defective area.

The scotoma occupying the centre of the field of vision cannot easily be demonstrated in the ordinary way until the acuteness of vision is so blunted that the patient cannot make out J. 16, then it is found that within an oval area stretching from the papilla to slightly beyond the macula lutea, a white object, especially if the examination be made in subdued light, does not appear so distinct as when seen by the neighbouring parts of the retina. The long axis of the oval scotoma is horizontal, and its point of greatest saturation somewhere between the nerve and centre. Sometimes indeed it may extend further horizontally, more of the nasal part of the field being involved. In all cases, except those in which the central scotoma is the first subjective manifestation of a progressive atrophy, the periphery of the field of vision remains intact, even although the central vision should sink so low as only to be capable of distinguishing light from darkness. The peripheral colour sense is also retained as a rule, although in some severe cases it is defective; but it is never

* *Loc. cit.*, p. 215.

so completely abnormal as in the region of the scotoma. Generally, it appears to be the nasal portion of the field where the perception of green and red hues is deficient, whilst immediately above and below the scotoma it remains normally acute. Leber and Nettleship have described cases in which the peripheral colour defect has taken the form of a ring-shaped scotoma.

In the less severe cases the examination of the colour-sense affords an easier and more trustworthy test of the presence of a scotoma. Within the abnormal area there is more or less red-green blindness. Therefore, all that is necessary practically in order to determine the existence of a central scotoma to account for a certain amount of amblyopia, is to find out whether a red or a green hue is seen more distinctly outside than within the bounds of an oval area occupying the position indicated above. This is simple enough, and yet it requires some care and practice, especially if the amblyopia be slight and the patient inattentive or stupid. Each eye should be examined separately. A small piece of red paper not larger than 10 mm. square should be used. This, if held at the end of a piece of whalebone, can be brought opposite any portion of the retina whilst the patient is steadily fixing some definite object large enough and bright enough to be easily seen. If the case be pronounced, the red will appear bluish, yellowish, buff, brown, white, or of an indefinite dirty colour, according to the hue and shade taken, and the part of the scotoma on which it falls. Small green objects will give rise to much the same sensations, but it is not necessary to use more than one colour, and different hues of red are, perhaps, more easily obtainable. The colours appear brighter in shade at the very centre than between it and the blind spot, where they are often described as dirty or smudgy, and it is in this latter region that the first evidence or last traces of a colour-scotoma can be detected, and indeed the macula does not always appear to be involved. If the amblyopia be less pronounced, we should often fail to detect the scotoma if we were merely to rely upon the naming of the coloured object

by the patient, he might call it red or green whenever it chanced to fall on the retina. In such cases it is well to use two different reds, one of a somewhat bluish and the other of a somewhat yellowish hue. By bringing these alternately 8° or 9° on either side of the point of fixation, as recommended by Krenchel, it will often be found that the red appears more distinct to the nasal side, being paler, more bluish, or more yellowish-red to the temporal side. Another plan which I frequently adopt, and which is still more delicate, is to use different shades of the same red by mixing with white; the palest is then a faint pink, and with these and an average amount of intelligence on the part of the patient, the very smallest defect can readily be detected.

The scotoma is entirely negative; thus the patient on looking at a large uniformly red surface, as he recognises the colour from the impressions received by the normal portions of his retina believes he sees it red throughout, and is entirely unconscious of the altered impression caused by the scotoma in the same way as we are unaware of the want of continuity existing in our field of vision over the surface occupied by the entrance of the optic nerve. For this reason, and owing to the circumstance that the colour-sense defect in the scotoma is of the same nature as in congenital colour blindness, so far at least as there is no blindness for one colour hue without at the same time a corresponding blindness for its complement, it is possible to choose two or more colours which give the same sensation at some particular point in the scotoma. And if one of these colours be given to a surface occupying a large portion of the field, whilst only a small portion of the surface opposite this particular point in the scotoma is of one of the other colours, the impression received is the same as if the whole surface were of uniform colour. By taking advantage of this, we have another good practical way of demonstrating the existence of a colour defect which involves direct vision, namely, by the use of Pflüger's colour tests, which are based on the principle of spontaneous contrast. Thus, in five cases in which the amblyopia was not

excessive,* I have found that none of the letters on the second sheet (red) could be made out when covered with one layer of tissue paper; the sheet appeared of a faint uniform colour, like pink blotting-paper. When, however, the attention of these patients was called to the presence of letters on the paper before them, especially after they had seen those induced from the yellow and blue and from most of the greens, there appeared to be a very faint induction from the peripheral parts of the retinæ, though not sufficiently distinct to enable them to read the letters eccentrically.

As to the ophthalmoscopic appearances, I think a little too much has been made of the paleness of the temporal half of the disc. This is by no means a constant symptom, nor does it appear to stand in any relation to the duration of the amblyopia. I have recently seen a case which had lasted two and a-half years, in which the discs presented a perfectly normal appearance both in respect to transparency and colour; a certain amount of pallor is certainly frequently met with in these cases, but as much is often found without any subjective symptoms whatever, and falls quite within the bounds of physiological variation. Further, it is a well-known fact that any paleness of the discs is always most marked on the temporal side. A shallow and broad physiological cup, by no means uncommon, gives a whitish aspect to this part of the disc, and in the case where the contrast between the two halves is marked, such a condition may coexist with a slight pallor associated with the amblyopia. At any rate this appearance is in no degree inconsistent with perfect recovery, so that we cannot ascribe to it any importance in the prognosis. Of more importance, however, is an appearance occasionally met with of a sort of insidious neuritis, a local perivascularitis associated with loss of transparency and

* As I have only just procured Pflüger's tests, I have not had an opportunity of trying them oftener, but the result in the cases tried is so consistent with what we know of the nature of the affection under consideration, that I have little doubt of their applicability to all cases where the colour scotoma involves the centre.

haziness of the disc; white streaks accompanying some of the vessels just as they emerge from the nerve, and not extending much, if at all, beyond the margin of the disc, sometimes they seem to bridge over the space from one vessel to another: in short, they are the evident signs of inflammatory change. This condition is only found in the severer cases. In the great majority of cases there is absolutely no pathological change to be found with the ophthalmoscope.

In the diagnosis of central amblyopia, the presence or absence of the colour scotoma is of primary importance, should the defect of vision be slight, or any detectable complication coexist either on the part of the dioptric media, retina, or choroid. But as a general rule, the symptoms are so evident, that this test is unnecessary to establish the diagnosis; still it is well to make it a matter of routine, and with very little practice this can be done without loss of time.

The prognosis is always good as far as complete blindness goes, except in the rare cases before referred to, where progressive atrophy begins by manifesting itself as a central scotoma. A case, probably of this nature, is at present under observation.

William R., æt. 43, gave a distinct history of tobacco amblyopia twelve years ago, from which he recovered in three months, on entirely leaving off smoking. After which he "saw as well as he had ever done." On March 10th, 1880, he applied at the Hospital with much the same symptoms as had characterised his previous amblyopia. He smoked $\frac{1}{2}$ oz. of shag daily, and stated that tobacco had never agreed with him. He did not exhibit any trace of alcoholism. V = fingers at 15′ with right eye; at 8′ with left; field normal, papillæ slightly hazy, marked central scotoma for red; also defective colour vision over the whole field. On April 28th, 1880, V, left as before; right slightly improved. Both fields of vision concentrically limited, left up to 10° from point of fixation above. He had not, according to his own statement, smoked more than $\frac{1}{4}$ oz. weekly since last visit.

The cases which remain stationary, or in which the central vision continues to get worse, are generally those which have lasted some time, in which there are decided ophthalmoscopic changes, and where alcohol forms a very suspicious element, possibly also there may be in such cases some other cause which remains undetected. In the typical form where there are no distinct pathological changes to be seen with the ophthalmoscope, improvement is certain to take place on the cessation of smoking. Generally it goes the length of complete recovery, but this may take many months or even years, although I have seen vision improve from $\frac{20}{70}$ to $\frac{20}{20}$ in less than a fortnight. Mr. Hutchinson has published cases of complete blindness which he ascribes to tobacco; the fact that the subjects in question were great smokers is not enough to render this even probable, besides in two cases the discs are described as being very white.

The treatment consists in withholding tobacco and diminishing if necessary the amount of alcohol consumed. This must be insisted upon. The patients will probably also require some "mixture" to which to ascribe their cure, the nature of which may be left to the discretion of the surgeon; few drugs of the Pharmacopœia will do harm.

Little can be said with respect to the seat of the lesion in central amblyopia. Of the numerous hypotheses which have been advanced, only those of v. Graefe and of Leber appear to claim attention. According to Leber, the central scotoma is caused by a partial neuritis or atrophy of the optic nerve, or it may be merely some anomaly in the blood supply to the superficial layers of nerve fibres. Graefe on the other hand, was of opinion that the symptoms could only be explained by the assumption of some more central disturbance. In support of this latter view, we have the symmetrical distribution and equal degree of the scotoma in both eyes, the absence in most cases of any pathological changes in the retina or optic nerve, the nature of the colour blindness, and we may, perhaps, add the frequent toxic etiology of the amblyopia, as well as its curative nature. Leber's view is supported more

by the anomalous cases which further experience may prove really to belong to another category. At any rate it is by no means improbable that much the same symptoms may be produced both by a peripheral and a central lesion.

ON A DESCRIPTION OF A NEW OPHTHALMOSCOPE.

By John Couper, M.D., F.R.C.S.,

Surgeon to the Royal London Ophthalmic Hospital.

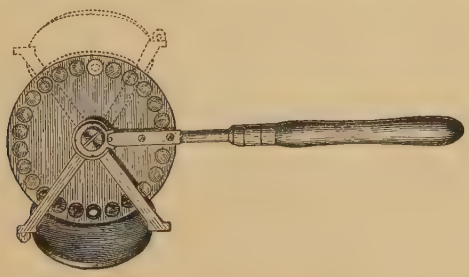
During the last few years I have modified the ophthalmoscope from time to time with a view to increase its accuracy and convenience in practice. It is unnecessary to specify the steps by which I have arrived at that form which I now present for consideration. In general terms my aims have been—1st. The utmost simplicity of parts consistent with efficiency, both as an ophthalmoscope and as an optometer; and 2nd. To avoid change of mirror or of disc, and in fact to dispense with all readjustment entailing trouble during an examination in a darkened room. Like other ophthalmoscopes aiming at completeness, it consists essentially of a concave mirror of silvered glass with a disc of lenses revolving behind it. Its chief distinctive features are—1st. The contrivance by which one mirror is made to serve all purposes, both of direct and indirect examination; and 2nd. The mechanism, by means of which each lens is so placed during direct examination that the observer looks in its principal axis.

It is superfluous to insist here on the disadvantage of creating astigmatism by an oblique position of the lens in regard to the axis of vision. The definition of the erect image is thereby diminished and exact optometry is impracticable. This defect is inevitable in the older ophthalmoscopes in which disc and mirror are fixed in a parallel position. It is obviated in some modern instruments by providing for direct examination a small special mirror fixed on the obliquely cut extremity of a short tube, and which can be placed at the angle necessary to reflect light into either eye by simple rotation of the tube. There are two objections to this plan—1st. It involves a change of mirrors. 2nd. It necessitates undue distance of the lens from the sighthole. In my instru-

ment the axis of the hinge which connects the mirror with the bracket is a tangent to the edge of the disc on the one hand, and on the other to the back of the mirror at a point close to the edge of the sighthole. The mirror is bare at this spot, in order to minimise the distance of the lens from the sighthole. Thus, when inclined at an angle with the disc, the mirror pivots on the edge of the disc, but without actual contact therewith during direct examination. On the other hand, it is made to stand parallel with the latter during examination of the inverted image by simply unbending the hinge.

By this arrangement the least practicable distance of lens from sighthole, and consequently of eye from eye, is secured. It is thus possible to use lenses of small size (of 5·0 mm. effective diameter) without optical inconvenience. A larger number of them can be set in one disc, and there is a gain in compactness. Obviously a tangent hinge alone ensures due proximity of lens to sighthole. While a hinge, the axis of which is a radius both of disc and mirror, necessitates a certain interspace between lens and sighthole which is by no means insignificant, even though the sides of the mirror be cut off to admit of sufficient tilting.

To adapt the instrument for direct examination of either eye the bracket carrying the mirror is made to describe a half circle round the disc. The mirror can thus be brought into play on either side of the disc at pleasure. This movement, which is of a simple kind, is provided with a stop-action which arrests the mirror at three several positions, viz.:—(*a*) At a point diametrically opposite the handle when the indirect method is to be employed. (*b*) On either side of the disc, as indicated by the dotted lines in the woodcut,



according as the right or the left eye is to be examined directly.

A glance at the drawing shows that during direct examination the disc must project in an outward direction considerably beyond the observer's orbit. Therefore, to avoid contact with the patient's nose, his eye must be approached from the temporal side, not from the front. All difficulties from this source are avoided by the ophthalmoscopist placing himself at the side, instead of in front, of the patient: by making the latter look outward, while he himself does the same, and by arranging that the respective heads shall be slightly inclined towards each other.

The mirror has a focal length of 8 inches and a diameter of 29·5 mm. It is no thicker than gives security against bending of the glass. The sighthole is central. Its bore is conical, the wider end being towards the observer's eye, which thus looks through a knife-edged aperture and not through a canal. The sides of this aperture have such a divergent slope that they do not reflect rays from the lamp flame into the observer's eye, and the appearance of a luminous haze, overspreading the sighthole and greatly disturbing vision, is thus avoided. With parallel sides the sighthole becomes a canal, the reflections from which may blot out the image, especially if the mirror be somewhat thick. With a conical sighthole a *thin* mirror is desirable, because the divergent sides being coated with lampblack must encroach unduly on the silvered reflecting surface if the mirror be thick. A mirror faulty in this respect is obviously valueless for examination through an unusually small pupil, only that part of its surface corresponding to the blackened sides of the bore would then reflect light into the examined eye. The thickness of the coated mirror should not much exceed 0·3 mm. The sides of the bore may then enclose an angle of 25° without any appreciable encroachment of the blackened on the silvered surface.

The diameter of the sighthole should not be less than 2·01 mm., otherwise there is stenopœic action, circles of dis-

persion are cut off, a comparatively defined image is obtained with a glass which does not measure the ametropia, and an erroneous estimate of refraction is made. Inaccuracies from this source are undoubtedly precluded by using a sighthole of 2·5 mm., and such an instrument may conveniently be used in cases where a mydriatic has been employed to dilate the pupil. It is inadmissible, however, in the case of a small pupil. With the smaller sighthole of 2·01 mm. diameter all stenopœic error is avoided if the mirror be held close to the cornea of the ophthalmoscopist.

Little need be said as to the lens series employed. The disc, which has a diameter of 41·5 mm., has 25 apertures, thus admitting of 12 concave and 12 convex lenses. For an emmetrope the following in dioptres will be found convenient:—

— 1	2	3	4	6	8	10	12	14	18	37	74
+ 1	2	3	4	5	6	7	8	10	12	14	18

A myope of course requires a different series. He must enrich the concave at the expense of the convex group. He needs more concave glasses than the emmetrope, because, in the direct method, his own myopia, as well as that of the patient, must be corrected by means of the lens behind the mirror. On the other hand, he requires fewer convex lenses the greater his myopia, because the latter is employed to neutralise the hypermetropia of his patient. Generally speaking, he should contract one series and enlarge the other by as many lenses as he has dioptres of myopia. In a similar way the hypermetrope who constantly wears glasses which absolutely correct his refraction, and who, therefore, has no more difficulty than an emmetrope in holding accommodation in abeyance during the use of the ophthalmoscope, must make a converse change. His convex series must be enriched at the cost of the concave. He needs more convex lenses than the emmetrope, because his own, as well as the patient's, hypermetropia must be corrected by the ophthalmoscope, and he needs fewer concave glasses, because

in the direct examination of the myopic fundus his hypermetropia is made to serve as a measure of the myopia of the patient.

The disc has a stop action which centres each lens with the sighthole. It acts lightly enough to admit of lens after lens being brought into play by pressure of the finger on the edge of the disc without breaking the continuity of an examination. The time necessary for estimating refraction is thus materially abridged.

I have found the higher concave glasses of great practical value in high grades of myopia. Strictly speaking, the use of such glasses in the direct method is impracticable, except under two conditions, viz.:—1st. The utmost approximation of the eyes concerned; and 2nd. The observer looking in the principal axis of the lens. The size and beauty of the image, together with the perfect definition of the fine pathological changes but too often present in such cases, attest the optical correctness of the mechanism by which these conditions are fulfilled. I have thus seen the region of the macula in a myope who required, for full correction, a minus lens of $1\frac{1}{3}$ inches. Those accustomed to rely on the ophthalmoscope as an optometer and to prescribe in appropriate cases glasses from its data, will find 24 lenses too few for this purpose. To obtain a larger series in the most convenient form I have had a twin ophthalmoscope constructed which consists of two such instruments as that just described. One has only convex the other only concave lenses in the disc. The numbers are coloured red in one, white in the other disc, so that it is easy to distinguish one half of the instrument from the other in the ophthalmoscope room. By this plan a total of 48 lenses is secured, viz.:—

− 0·5, 1·0, 1·5, 2·0, 2·5, 3·0, 3·5, 4·0, 4·5, 5·0, 5·5, 6·0, 6·5, 7·0, 7·5, 8·0, 9·0, 10·0, 12·0, 14·0, 16·0, 20·0, 37·0, 50·0

+ 0·5, 1·0, 1·5, 2·0, 2·5, 3·0, 3·5, 4·0, 4·5, 5·0, 5·5, 6·0, 6·5, 7·0, 7·5, 8·0, 8·5, 9·0, 9·5, 10·0, 12·0, 14·0, 16·0, 18·0.

This series is ample enough for all purposes of exact

optometry. Each power is obtained from a single lens, an arrangement which has optical advantages over that which obtains certain powers by the superposition of lenses. My friend, Mr. Adams Frost, has added to the single instrument a small straight slide bar bearing three lenses, each of which may in turn be added to any in the disc itself. By this method no doubt an extended series of both + and − glasses can be obtained from one disc. The comparative cheapness of this plan is all that can be urged in its favour.

Under the best conditions yet devised the illumination of the fundus is comparatively feeble. If we needlessly interpose a stratum of glass with two refracting surfaces, neither of which is wholly free from optical flaws, the definition and brilliancy of the image will, *cæteris paribus*, be impaired. For precisely the same reason it is better that the mirror should have a well-constructed sighthole rather than a central spot, from which the silver has been erased, and these instruments give least definition and the dimmest image which have no perforated sighthole and with which it may chance that the rays emerging from the patient's eye encounter three strata of glass and six refracting surfaces, with the optical imperfections incidental to them, before reaching the eye of the observer.

Messrs. Pickard and Curry, 195, Great Portland Street, are the makers of the instrument. I am glad to be able to acknowledge the ingenuity and thoroughness with which they have embodied my design. The accuracy of their workmanship leaves nothing to be desired, and they produce the instrument at a very moderate cost.

ON KERATOSCOPY.

By Litton Forbes, M.A., M.D. Pennsylvania,
Clinical Assistant at the Hospital.

Under the above title, Prof. Cuignet, of Lille, some years ago (1874) called attention to a method of determining the refraction of any eye by simple inspection of its cornea with transmitted light. For this purpose he made use of an ordinary ophthalmoscopic mirror, held at a definite distance from the eye under observation. The mirror was then rotated in various directions round its own axis, and the movements of the resulting lights and shadows upon the cornea were noted. From the general appearance of the bright pupillary reflex, and from the direction in which its accompanying shadow moved as compared with the movements of the mirror, M. Cuignet was enabled with more or less accuracy to diagnose at once not only the nature but also the degree of any ametropia which might be present. He was the first to systematise this method, and to show how far it might be made serviceable in suitable cases. Ten years previously, however, Mr. Bowman had already called attention "to the possibility of diagnosing regular astigmatism by using the mirror of the ophthalmoscope much in the same way as for slight degrees of conical cornea."* This suggestion would not, however, appear to have attracted the attention it deserved, and both Mr. Bowman's remark and M. Cuignet's more elaborate precepts, subsequently fell more or less into oblivion. Of late, however, the subject of keratoscopy has once more attracted attention, especially in France, while, so far as I am aware, it has not yet been touched upon by any English writer.

For some time past I have been accustomed to use the ophthalmoscopic mirror only in the diagnosis and estimation

* Donders, "Accommodation and Refraction of the Eye," English translation, p. 490.

of the degree of ametropia. The method has much to recommend it, with certain limitations and exceptions to be mentioned hereafter. Meanwhile I think it will be possible to demonstrate the following propositions with regard to keratoscopy :—

1. The existence of emmetropia, myopia, or hypermetropia, or the simultaneous presence of any two of them in different degrees in the same eye (astigmatism) may be diagnosed by simple inspection of the corneal reflex.

2. This reflex, as also the movement of the light and shade on the cornea when the mirror is moved round on its own axis, depends in low degrees of ametropia almost entirely upon the distance of the observer from the observed eye. Moreover, these appearances will vary according as a concave or a plain mirror be used.

3. The term keratoscopy is a misnomer, the form of the cornea having little or nothing to do with the resulting phenomena. These depend mainly upon the position of the retina with regard to the focal length of the dioptric system of the examined eye.

4. This method of inspection affords no means of distinguishing between axile ametropia and ametropia of curvature.

Before proceeding to consider in detail the various appearances presented by an illuminated pupil under certain conditions of refraction, a few words may be said as to the best method of making this examination. The main object should be to secure a well marked contrast between the illuminated and non-illuminated portions of the pupil, and this can, perhaps, best be done by means of a screen so arranged as to shut off all rays of light proceeding directly from the lamp to the eye under examination. The chamber in which the examination is conducted should also be as dark as circumstances will allow, inasmuch as nothing masks the phenomena of keratoscopy so much as a dull diffused light. The mirror, if a concave one, should not be held at its focal distance from the examined eye, as a small bright

circle of diffusion would thereby be produced on the retina, with the result of inducing contraction of the pupil. Supposing the mirror to be 20 centimetres focal length, it will be held most conveniently at about 50 centimetres from the patient. It should not be in the direct line of his corneal axis, but at an angle of about 15 degrees to one side of it, so as to bring the optic disc within the line of sight. In this way a more brilliant reflex will be obtained, while in cases where a mydriatic has not been used the pupil will be less liable to contract. The mirror should also be inclined at small angles, and rapidly moved through the various meridians of the cornea. This is especially important in cases of astigmatism. In low degrees of ametropia a mirror of shorter focus, say 10 or 15 centimetres, may occasionally be used with advantage. On the whole, however, it is perhaps better to use, on all occasions, the same mirror under identical conditions of distance and illumination. In this way only will it be possible to perceive those minute variations upon which the diagnosis of the degree of ametropia so much depends.

I shall now examine in detail the appearances presented by (1) the emmetropic, (2) the myopic, (3) the hypermetropic, (4) the astigmatic eye. My account of these will agree in the main with that given by MM. Cuignet and Mengin. In some important points, however, my own observations do not coincide with theirs.

If the fundus of an emmetropic eye be illuminated with light passing directly through the axis of the cornea, the following appearances will be observed. A dull red reflex will be visible over the whole field of the pupil; in the centre, however, of the field, the reflex will be somewhat dark, while around its circumference it will appear brighter and better marked. If the cone of light be next allowed to fall obliquely upon the cornea, the following changes will be noticed. A shadow which, roughly, has somewhat the form of a triangle, with its base resting almost on the pupillary edge, and its apex extending beyond the centre of the cornea, will

occupy one half of the illuminated disc of the pupil. If, for example, the light be directed upon the external portion of the right cornea, the shadow will be perceived upon the same right side, and its shape will be that of the triangle just described. This triangle will have its base directed towards the external portion of the cornea, but will not reach quite so far as the periphery. Between its base and the portion of cornea corresponding to the pupillary margin there will be a clear bright space. The apex of the triangle will be directed towards the centre of the cornea, the horizontal meridian of which might be said to bisect this angle. The remainder of the cornea will be brilliantly illuminated. If now the luminous disc formed by the reflex of the mirror be directed towards the nasal side of the cornea, the shadow will be seen to move in the opposite direction, and the same will be true according as the superior or inferior portion of the cornea be illuminated. Thus if the light be allowed to fall upon each quadrant of the cornea, by turns, the same relative position of light and shade will always obtain.

In the emmetropic eye, therefore, the shadow occupies the centre of the cornea, and extends more or less into the illuminated half. In whatever direction the mirror (concave) is moved the shadow will follow its movements. These appearances are constant in all emmetropic eyes. If distinctly seen in every meridian their presence precludes the existence of moderate or high degrees of myopia, hypermetropia, or astigmatism.

2. In the myopic eye the appearances are essentially different from those just described, and in high degrees are very characteristic. In this form of ametropia one portion of the cornea is illuminated, while the other is plunged in shade. The amount of surface, so to speak, occupied by the illuminated portion will vary directly as the degree of myopia, being always greatest in high degrees. The shadow consequently in such a case will be proportionably small in extent, and moreover will follow the movements of the mirror instead of travelling in an opposite direction. If the

mirror be turned round its horizontal axis, the luminous triangle will describe a corresponding circle around the cornea. In high degrees of myopia the apex of this luminous triangle extends considerably beyond the centre of the cornea. The shadow is, therefore, seen as a small peripheral crescent situated at the opposite edge of the pupil. In moderate degrees of myopia the bright reflex does not extend so far as the centre of the cornea. It does not, therefore, include the apex, but nevertheless occupies an appreciable portion of the illuminated pupil. In low degrees of myopia I am very sceptical, for reasons to be given further on, of the diagnostic value of keratoscopy. It is, so far as my experience goes almost impossible to distinguish such cases from low degrees of hypermetropia or from emmetropia. In describing the appearances presented by slightly myopic eyes, M. Mengin speaks of the reflex occupying but a very small surface of the pupillary edge, and assuming the form of a thin crescent placed alongside the luminous portion, and turning with it in response to the movements of the mirror; but of the accuracy of this description I have not been able hitherto to satisfy myself.

3. In the hypermetropic eye, under the condition of examination already stated, one portion of the pupillary field appears brilliantly illuminated, while the other lies in comparative shade. The shaded portion occupies the periphery of the pupil, towards the side from which the light comes. This shadow will move regularly from side to side in each meridian, or will circle round the pupil in accordance with the movements of the mirror. These movements it follows, instead of, as was the case in myopia, travelling in an opposite direction. As regards the degree of hypermetropia, it may be said generally that in low degrees the shadow occupies half or even more of the corneal surface, and has the form of a triangle, with its base at the periphery and its apex at the summit of the cornea. In moderate degrees it occupies a smaller surface, and extends scarcely so far as the summit of the cornea, assuming a distinctly crescent shape.

In very high degrees of hypermetropia it takes the form of an extremely thin crescent, deeply shaded, and surrounding the edge of the pupillary field. The hypermetropic eye is, therefore, characterised by appearances exactly the reverse of those of the myopic. In the latter the *bright portion* is situated at the periphery, on the side from which the light comes, and increases in extent in proportion to the degree of myopia. In hypermetropia the *shadow* occupies the above position, and diminishes in extent in proportion to the amount of hypermetropia present. From the intensity of the light and shade, and the contrast between them, an approximate estimate of the degree in which hypermetropia or myopia is present may be formed.

4. *Astigmatism.*—In this anomaly of refraction, as a rule, one or more meridians of the cornea are either hypermetropic or myopic, but in unequal degrees. Cases of regular astigmatism are therefore classed according as one or other form of ametropia preponderates. In mixed astigmatism both forms of ametropia are present in the same eye, though not necessarily in equal degrees. Simple astigmatism, whether myopic or hypermetropic, and especially if at all considerable, may be very readily diagnosed by means of keratoscopy. Take first a case of (Am) simple myopic astigmatism, in which the vertical meridian is myopic. Examining successively the internal and external halves of the cornea with the concave mirror, the shadow will be seen to occupy successively the same position as in a normal cornea, and its movements will not coincide with those of the mirror. Examining in the same way the vertical meridian, that portion of the cornea will be illuminated which lies towards the direction whence the light comes, and the shadow which corresponds to it will travel in the same direction as the movements of the mirror. The illuminated portion will assume the form of a crescent, or of a triangle, with its apex towards the summit of the cornea, and will be large in proportion to the degree in which myopia is present. If the crescent is small, the myopia is probably low in degree, but if it occupies half of the cornea,

or even more, in all probability at least 3 D will be required for correction. In compound myopic astigmatism (M + Am) the general appearances in each meridian will be the same so far as regards the direction in which the shadow moves, but they will differ in degree. Thus in both meridians the shade will move in the same direction as the mirror, but if the vertical meridian is more myopic than the horizontal, the illuminated portion of the pupil will be relatively larger in the vertical than the horizontal meridian. Similarly in simple hypermetropic astigmatism, the shadow will in both meridians move in a direction contrary to the movements of the mirror. In the hypermetropic meridian it will lie well at the periphery, and will be more pronounced in proportion to the amount of hypermetropia present. In compound hypermetropic astigmatism the shadow will be smaller and deeper in that meridian in which the anomaly of refraction is the most marked. In mixed astigmatism the movements and arrangement of the light and shade will vary in each meridian according to the kind and degree of ammetropia present. As regards irregular astigmatism, I have not been able to observe any constant appearances. The play of light and shade is irregular and fantastic, and does not admit of accurate analysis. The following case of regular astigmatism occurring in the practice of Mr. Streatfeild, will serve to illustrate the value of keratoscopy as an aid towards forming a rapid and approximately accurate diagnosis.

Case.—Caroline D., seamstress, aged 22, complained of difficulty in working and reading, the eyes being always more or less painful and fatigued. She volunteered the statement that she saw vertical lines better than horizontal. V in each eye without glasses $= \frac{20}{200}$.

In the right eye in the vertical meridian the shadow caused by illuminating the pupil with a concave mirror held at the distance of 2 feet, followed the movements of the mirror. The illuminated portion of the pupil was in the form of a triangle, the apex of which extended beyond the summit of the cornea.

In the horizontal meridian the shadow likewise moved in the same direction as the mirror, but was larger and not so deep as in the vertical. In the left eye the illuminated portion was fairly bright in each meridian, and occupied a large portion of the field of the pupil; the shadow here also followed the movements of the mirror. From these appearances a rough diagnosis was at once made of a considerable degree of myopia in both eyes with compound myopic astigmatism in the right, the vertical meridian being more myopic than the horizontal. Atropine was not used. With the ophthalmoscope I found for the vertical meridian in the right eye M = 4 D, for the horizontal M = 2 D. Tested for distance by means of a stenopaic slit the same results were obtained.

Vision in the right eye with spherical glasses was brought up to $\frac{20}{40}$, and with cylinders to $\frac{20}{20}$. In the left eye I found by the same methods that the vertical meridian had M = 4 D, while the horizontal had M = less than 5 D. With a spherical lens — 4 D, and a cylinder, — 0·50 D, axis vertical, V = $\frac{20}{20}$.

Did space permit other and similar cases might be recorded, in which an approximately correct diagnosis was arrived at by the same method.

When a plane mirror is used instead of a concave the resulting phenomena are somewhat different. With a plane mirror the relative positions of the bright reflex and its corresponding shadow are transposed, and therefore in emmetropia, hypermetropia, and low degrees of myopia, the movements of the corneal shadow will be in the same direction as those of the plane mirror. If the mirror be pivoted from left to right the shadow will also move from left to right. On the contrary, in moderate and high degrees of myopia it will travel in a direction opposed to the movements of the mirror. The play of light and shade obtained therefore with a plane mirror and a myopic eye will, as regards the position and movements of the shadow, be precisely the same as those witnessed in a hypermetropic eye when a concave mirror is used. The truth of this assertion can easily be demonstrated

experimentally. Theoretically, it depends upon the fact that rays of light proceeding from a plane mirror are divergent, while from a concave they can become divergent only after previous convergence and intersection. Rays of light proceeding from any mirror are necessarily refracted in their passage through the dioptric media of the eye, and go to form an inverted image of any luminous point on the retina. With a concave mirror, however, this inverted image is seen only in cases of emmetropia and hypermetropia, or very low degrees of myopia. In high degrees of myopia the inverted image becomes inverted a second time, and thus appears upright to the observer.

The play of light and shade, and the apparent movements of a shadow occupying any portion of an illuminated pupil, depend mainly in the degrees of myopia upon the distance at which the observer is placed. This may easily be demonstrated experimentally in the following manner, as suggested by M. Parent. If a glass of + 1 D be placed before an eye already known to be emmetropic, and it be thereby rendered slightly myopic, an observer at a distance of say 75 centimetres will perceive the corneal shadow moving in an opposite direction to the movements of his concave mirror. This shadow will, however, be very feeble, and will require, in order to be satisfactorily observed, a mirror of a small radius of curvature, in other words, a large circle of diffusion, together with a weak illumination, and the effect of contrast in a well-darkened room.

In an eye myopic to the extent of 1 D a real and inverted image of its fundus will be formed at the distance of 1 metre. If the observer be at a distance of 40 or 50 centimetres from such an eye he will be within the focus of its dioptric system, and will see the circle of retinal illumination in the upright image. The shadow upon the cornea will therefore travel in an opposite direction to the movements of the mirror, just as in emmetropia or hypermetropia. In proportion also as he withdraws further from the observed eye he will receive rays ever increasingly convergent. When he is at 80 centimetres

from the nodal point of this eye the convergent rays will be at not more than 20 centimetres from their point of intersection. Under such circumstances if the observer is emmetropic they will meet in front of his retina, and will form a circle of diffusion equal to that which would be formed in the case of a myopic eye of 5 D, which should fix an object placed at the distance of 1 metre. At a still greater distance, say 95 centimetres, the rays of light are only 5 centimetres from their point of convergence. The circles of diffusion are consequently still further increased, and the condition of his vision will, under the circumstances, be similar to that of an emmetropic person who attempts to see objects situated at the distance of 1 metre with a glass +20 D before his eye. If the observer gradually withdraw to a still greater distance, the circle of diffusion upon his retina will continue to increase until, at a given moment, the real and inverted image of the illuminated circle will lie between the cornea and anterior focus of his eye, and the circles of diffusion will then have attained their maximum. Under such conditions all gradations of light and shade will be imperceptible. Nothing indeed will be visible but a uniformly illuminated circle, projected to a certain distance, and affording no keratoscopical indications whatever. Let a printed page be looked at under similar conditions, and its surface will appear of a uniform grey, the lines and interspaces forming one blurred and confused image.

If, however, the observer withdraws to a distance greater than 1 metre from an eye myopic = 1 D, he will perceive a shaded and illuminated portion when he has reached a distance of 115 centimetres, or sooner or later according to his amplitude of accommodation. But under these circumstances the shadow will no longer move, as it did before, in a direction opposite to that of the mirror. It will now move in the same direction, and a real and inverted image of the illuminated retinal circle will be visible. In low degrees, therefore, of myopia the corneal shadow will move either in the same or an opposite direction to the movements of a concave mirror,

according to the distance at which the observer is placed. It follows from this that keratoscopy can be but of little service as a means of diagnosing slight degrees of myopia.

At distances greater than one metre the characteristic appearances are difficult to seize except under peculiarly favourable conditions. For in such slight forms of myopia, the reflex being seen in the upright image, while the rays of light from it are convergent, circles of diffusion are apt to be formed upon the observer's retina. The degree of enlargement moreover being greater in M than in H, the characteristic appearances of light and shade are necessarily spread over a larger surface, and must, therefore, be less intense.

Although the term "keratoscopy," as applied to the reflex obtained from illuminating an eye under the conditions already mentioned, may be retained for convenience sake, it is clearly a misnomer. The phenomena observed depend almost entirely upon the position of the retina with regard to the focus of the dioptric system of the eye examined. In myopia the retina lies beyond, in hypermetropia within, the focal length of this system. Now rays of light proceeding from a concave ophthalmoscopic mirror held at about 50 centimetres from the examined eye, will produce upon its retina an inverted image whatever be the conditions of refraction present. This image will therefore move in exactly an opposite direction to any movement executed by the mirror. In emmetropia we have an upright image of objects at the fundus of the observed eye. The image of the flame projected upon the retina will therefore be seen in its true position, viz., inverted, and its movements will be opposed to those of the mirror. The same is also true for hypermetropia. In myopia the retinal image is equally inverted, but when seen at a certain distance, namely, beyond the punctum remotum of the observed eye, it is a second time inverted by the observer's eye. The movements of the mirror are, therefore, really inverted twice, and, consequently, the motions of the flame, the shadow, and the mirror, are all apparently in the same direction. But as already remarked, the intensity

of the shadow, or what amounts to the same thing, the brilliancy of the illuminated circle, will be proportional to the amount of ametropia present. The reason of this is sufficiently evident. In hypermetropia we obtain an upright and virtual image of the circle of retinal illumination, which image is small in proportion to the amount of H present. In myopia we obtain a real and inverted image, and the higher the degree of M the smaller this inverted image will be. In keratoscopy we see the circle of illumination in H in the upright image, and in M in the inverted, and, consequently, in proportion to the amount of ametropia present, so will the circle of illumination and its shadow be correspondingly small. Thus, in the image of the retinal reflex which is half illuminated and half in shade, the light and shadow occupying respectively a smaller space must, in accordance with a well-known law, be more intense. The converse is also true, and therefore the more nearly emmetropic an eye is the less marked will be the movement and the contrast of light and shade. This may be demonstrated experimentally, as pointed out by M. Parent. With a biconvex lens of 7 centimetres focal distance let a printed page be looked at, the lens being held so as not to form a real and inverted image of the print. At the distance of 6 centimetres (H) the characters will appear black and well defined. At 7 centimetres (emmetropia) they will appear magnified, but scarcely so well defined as at the shorter distance. At 8 centimetres (M) from the page, the lens being held close to the observer's eye, the characters become blurred and are no longer black, but grey. If the distance be still further increased, the upright image being retained, the type will become so pale and confused as scarcely to be visible.

In keratoscopy, as already sufficiently shown, the position and movements of the corneal reflex are directly correlated with the refraction of the eye observed: they can, therefore, be so interpreted as to give, within certain limits, more or less accurate information as regards any anomaly of refraction which may be present. The indications afforded apply, how-

ever, only to the sum of the refraction of all the media. No clue whatever is furnished by which an anomaly of refraction could be localised in one component of the dioptric system rather than in another. Keratoscopy is, therefore, useless as a means of differentiating between axile ametropia and ametropia of curvature. It can at most inform us as to the position occupied by the retina in relation to the dioptric system as a whole. Its indications will, moreover, be precise and trustworthy, exactly in proportion as the retina lies markedly beyond or within the focal length of that system.

In conclusion, a few words may be said as to the value of keratoscopy from a purely practical point of view. My own experience of the method does not by any means enable me to agree with the views as to its value put forth by M. Cuignet and others. As a rough and ready method of diagnosing high degrees of ametropia, whether myopia, hypermetropia, or astigmatism, it is certainly both convenient and trustworthy. In low degrees of ametropia it is neither easy of application nor can the results it gives be at once accepted as absolutely correct. Its chief value lies in the readiness with which by its means cases of mixed astigmatism may be rapidly diagnosed. It has also the advantage of rendering an observer independent of the patient's statements ; but this it shares in common with the displacement of the retinal vessels and with other well-known methods of diagnosing refraction. In cases of nystagmus, in which the observation of the direct image is sometimes confessedly difficult, as also in cases of contracted pupil, keratoscopy will often be found to render good service. While, therefore neither very accurate nor of very wide application as a means of diagnosis, it nevertheless possesses a certain value when used in conjunction with other and more strictly scientific methods.

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PULSATION IN EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA.

By A. Stanford Morton, M.B.,

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In embolism of the central artery of the retina, the phenomenon of a current of blood moving slowly and uniformly, or in detached discs or columns with intervening light spaces and in a jerky manner, in the affected vessels, has been referred to by various authors, and may be observed in the greater number of cases. Though, however, I have seen each of the above movements in the arteries, I have never seen them as they have been described in the veins.

The jerky onward movement is probably due to an embolus only partially blocking the calibre of the vessel affected, so that small quantities of blood may be forced past the obstruction in an irregular manner, but not in sufficient amount to fill the collapsed vessel. The to-and-fro movement is not so easily explained in this way but might be produced by a collateral vessel lending a small supply of blood, which moves in either direction in the collapsed artery, until a small quantity, escaping from the central side of the obstruction, furnishes the impetus which hurries it onwards.

The peculiar, very slowly moving, uninterrupted, onward flow of variously mixed darker and lighter shades of blood may find a possible explanation in the supposition that when the central embolism becomes impacted a small fragment is detached and carried on to the distal side of the portion in view, where, by retarding the flow of even what little blood passes the primary embolus, it gives rise to a slow stagnant current and partial separation of corpuscles and liquor sanguinis. Where the blood cylinders come to a complete stand-still, as in the case mentioned by Soelberg Wells in his

"Treatise," the second embolism has probably been complete. In some cases on record the movement of the blood is said to have been "often for a short time pulsatile," and in one which came under my notice at the South London Ophthalmic Hospital last March, when seeing Mr. Watson's patients, this symptom presented itself in a remarkable degree. The patient, a man æt. 34, came complaining of having noticed his sight "cloudy" for a week, but on covering his right eye two days previously to his visit, he found that he could not see with the left, with which he now had bare perception of large objects to his temporal side. On examination with the ophthalmoscope, I found the usual appearances of embolism, such as œdema of the retina and cherry-red spot at the macula, but the most remarkable feature was the steadily rhythmical onward pulsatory jerks of the regular blood discs or cylinders with intermediate light spaces, into which the contents of both branches of the bifurcated central artery were divided, and though sometimes these coalesced and passed on irregularly, yet several times during the observation I was enabled, by steadily fixing my attention on one blood disc, to count eleven pulsations or onward jerks during the ten seconds which it occupied in traversing the distance from the point of entrance of the artery to the periphery of the optic disc, from which position, though continuing nearly to the periphery, it was difficult to follow the pulsations closely enough to count them.

On again seeing this case, ten days later, no current or spontaneous pulsation could be detected in any of the arteries, which were all evenly filled, of fair size, and showing pulsation on firmly pressing the globe. The œdema was rapidly lessening, and the vision then or soon after was Jaeger 12. In this case the embolism, though in the central artery, must have been partial and rapidly absorbed, or the collateral circulation speedily established.

A case is reported by Liebreich, where cylinders of blood were observed to flow in a jerky manner in the vessels over a tumour between the choroid and the retina, which has a

special interest in reference to the case I have noted above, inasmuch as in it there was albuminuria present, and it might be supposed that the appearances described were due to pressure on the vessels produced by albuminuric œdema, but the fact of the symptoms being unilateral, and the obstruction posterior to the entrance of the central artery, would negative this theory.

A SIMPLE MODE OF ASCERTAINING THE ACUTENESS OF VISION IN YOUNG CHILDREN.

By George Lawson, F.R.C.S.,
Surgeon to the Hospital.

All surgeons who have much to do with ophthalmic work must have experienced a difficulty in estimating the sharpness of vision in young children who do not yet know the alphabet, and whose sight cannot, therefore, be tested by any test types. I have lately used a very simple method, which seems to answer well. To quote a case :—

A young child, æt. 4 years, had a marked strabismus, and it was desirable to ascertain the sharpness of vision in each eye. I took a plate or a small *papier-mâché* tray, and sprinkled on it some mixed bird seeds, hemp, canary, millet, rape, flax, and maw seeds. I selected a hemp seed, and having covered up one eye with the hand, gave the seed to the child, and asked him to pick out a similar seed from those on the plate. When he had accomplished this task, I took a smaller seed, and then one still smaller, until at last I gave him a maw seed which will correspond as nearly as possible to a letter of Jæger 1 test types.

If the child can match the maw seed, we may fairly conclude that had he been able to read he could have read Jæger No. 1. Both eyes may, of course, be tested in the same manner. The distance at which the child holds his head from the plate whilst matching the seed given to him may also be taken into account.

Beads of different sizes would scarcely do as well, as it would be difficult for a child to compare the different sizes in beads which were all black, and coloured ones would not answer, as the child would be guided by the colour in his selection.

HOUSE SURGEON'S NOTES—TREATMENT OF IN-PATIENTS.

By R. Marcus Gunn, M.A., M.B,
Late Senior House-Surgeon.

Gonorrhœal Ophthalmia.

When one eye only is affected the sound eye is at once protected by the application of Buller's shield, kept in position by strips of strong adhesive plaster.*

Treatment of the Inflamed Eye.—Iced compress or iced alum lotion is kept constantly applied over the lids, and a solution of chloride of zinc (2 grs. to the ounce) dropped into the eye from three to six times in the twenty-four hours, the frequency varying according to the amount of the discharge. The everted lids are painted once daily with a solution of nitrate of silver (20 grs. to the ounce), and the patient is instructed to bathe the eye frequently with cold alum lotion. He takes the ordinary hospital diet with the addition of tonics when the general health demands it, and the urethral discharge is treated locally.

Under this treatment the conjunctival discharge is seldom very troublesome; but, should it collect much under the lids, syringing with cold water two or three times daily is employed.

The manner of application of the iced compress will be described later, under the treatment of congenital cataract. The addition of a little ordinary alum to the melted ice constitutes the iced alum lotion.

Should there, on admission, be decided localised corneal haze, with or without ulceration, or should such a condition appear after admission (as it sometimes will notwithstanding all precautions), cold poppy lotion is used instead of the iced compress, and drops of a solution of sulphate of eserine (2 grs. to the ounce) are dropped into the eye six times daily. The chloride of zinc drops are often discontinued

* For description and method of application, *see* The Lancet, May 16, 1874. Also Lawson, "Diseases of the Eye," 4th edition, p. 10.

or a weaker solution is employed, but even in cases of deep ulceration this solution does not cause the irritation that might be expected. The brushing of the lids is continued, great care being employed in their eversion, so as to avoid rupturing the affected corneal tissue.

When the discharge has nearly ceased the lids are only brushed once every second or third day, and this treatment is gradually discontinued. The good effects of eserine on the true, deep gonorrhœal ulcer are very decided, and even where there is a large sloughing of cornea these drops may be used with great advantage.

"Buller's shield" mentioned above affords an excellent protection to the sound eye, but great care is needed in fixing the plaster at first; and the nurse is instructed to examine its security frequently, as the constant application of fluids to the affected eye is apt to loosen the strips fixed on the nose.

Should the patient complain of slight irritation of the sound eye, with gumming of the lids, *while the plaster is still properly fixed*, the shield is not at once removed, but the condition of the eye is observed repeatedly (as can readily be done through the watch-glass); if the inflammation increases and discharge begins to show itself, the shield is at once removed, and the eye treated energetically with iced lotion, &c., as above. Very frequently, however, the symptoms of irritation and gumming accompany the use of the shield, and do not give rise to any serious inconvenience.* In such cases it is best to raise the protector from the outer side, to bathe the eye with a clean rag and alum lotion, and to apply a little simple ointment to the edges of the lid: the protector is then replaced and fixed as before.

Corneal Ulcers.

The treatment of ulceration of the cornea varies necessarily according to the character of the ulcer. Where it is

* In these cases I have generally found an old history of weak eyes and gumming in the lashes. Such patients have been in the habit of rubbing their eyes in the morning and picking their lashes with their fingers, and this doubtless accounts for their liability to contract gonorrhœal ophthalmia.

deep, with an irregular, undermined periphery and surrounding infiltration, with or without hypopyon, nothing gives such good results as the use of eserine drops five or six times daily. The eye is at the same time bathed for some minutes every half hour with hot poppy fomentation, and quinine is administered internally thrice daily.

If there be much pain from the commencement, or if the eserine drops cause it to appear, bathing with hot belladonna fomentation and the instillation of atropine may be substituted; but in such cases the progress towards recovery is generally less rapid, and the result less satisfactory.

In cases of abscess of cornea, or if there be dense purulent infiltration surrounding an ulcer, a section right through the affected tissue is made with a linear knife, and the eserine treatment pursued. It is seldom necessary to re-open this wound, as in Saemisch's operation.

A seton in the temple is of benefit when there is persistent photophobia, and also in cases of vascular ulcer.

Stimulation with strong solution of nitrate of silver (applied to the part with a fine camel hair brush) is used in the indolent clear ulcer. Phlyctenular ulcers when they do not tend to perforate, and when they are not accompanied by much photophobia, are treated by insufflation of calomel, or the application of dilute yellow oxide of mercury ointment (Pagenstecher) once daily.

In all cases of ulceration a large shade is used, and the patients are kept in a darkened room.

Soft Cataract.

Immediately after needling a drop or two of atropine solution is put into the eye and an ice compress applied. The latter is kept on for the next two or three days, and is then removed if there be no pain or marked ciliary injection. Atropine (4 grs. to the ounce) is dropped in six times during the day and two or three times during the night for the first two days; it is then used about four times daily. The condition of the bowels is attended to.

Immediately on the occurrence of pain, or if the globe become tense, two leeches are applied to the corresponding temple, or an ounce of blood is removed by Heurteloup's artificial leech.

If the anterior chamber is well filled with soft lens matter, or if the eye continues too tense, the swollen lens is removed on the fourth day by suction-syringe (Teale's or Bowman's) or curette. After this operation the ice compress is re-applied, atropine used frequently, and blood abstracted from the temple if there is pain.

On removing the ice compress a dry pad is kept on and the eye well shaded.

Senile Cataract.

After the ordinary linear extraction a small strip of plaster is laid across the closed lids of each eye so as to prevent the possibility of the patient opening his eyes during the period of excitability after the anæsthetic. A small piece of lint and a circular pad of cotton wool are laid over each eye and secured by a cataract bandage.

At the evening visit this dressing is removed, the patient being warned not to attempt to open his eyes. Each strip of plaster is now raised from below and the lower lid gently drawn downwards so as to allow the escape of any pent-up tears which frequently collect and give rise to much discomfort. The strip of plaster is then replaced and dressing applied as before, fine linen being placed next the eyes. The condition of the bowels is inquired into, and a gentle aperient given if necessary.

Next morning the eyes are bathed gently with luke-warm water and the strip of plaster removed entirely. The lids are dried carefully with some absorbent cotton wool. The lower lid is now drawn down slightly as before and fresh dressings applied. Oil or simple ointment is rarely used for the lids, and that only when there is an eczematous condition, causing the dressing to adhere to them.

In the evening the dressings are again removed and fresh

ones applied, but there is no necessity for *bathing* the eyes at this time in ordinary cases.

This treatment is continued for the first week, when, if all has gone well, the case is put into the hands of an experienced nurse, who attends to the dressings in future. On the tenth day the cataract bandage is discontinued and the second eye left uncovered. Three or four days later both eyes are uncovered, a shade only being worn over.

The various accidents and contingencies of the after-course of the treatment are met as they arise.

Iritis, &c.—On the occurrence of *pain*, which cannot be accounted for by any bad position of the dressings, two leeches are at once applied to the temple. Iritis seldom appears before the fourth day, and one of its earliest symptoms is slight *itching* on the eye, oftener accompanied by a creeping sensation down the corresponding side of the nose. When such symptoms occur leeches should be applied *at once*.

Heurteloup's artificial leech is not to be recommended soon after cataract extraction, as it drags too much on the eyelids and may cause reopening of the newly-healed corneal wound.

Indications for the use of Atropine.—Atropine is used (1) in cases where lens-matter has been left in the eye at the time of operation; (2), when, from some difficulty in the extraction of the lens, unusual interference with the iris has been necessitated; (3), on the occurrence of symptoms of iritis. In the first two conditions the atropine instillation is commenced on the evening of the second day.

When slight iritis with lacrimation appears after the first week a blister to the temple or over the mastoid process is useful, and may be repeated every second day if necessary. Atropine sometimes causes irritation in such cases, and daturine (4 grs. to the ounce) should be substituted; or, if a stronger mydriatic is called for, duboisine (1 gr. to the ounce) may be used.

Suppuration of the edges of the wound generally first shows on the morning or evening of the second day, and is recognised by œdema of the lids with a slight purulent discharge on the dressing. Frequent bathing with hot poppy fomenta-

tion is now required and eserine should be dropped into the eye thrice daily. The bowels are freely opened by colocynth and mercury pill and afterwards quinine or ammonia with bark is administered. Leeches are applied if much pain be present. Under this treatment the corneal suppuration seldom runs a very severe course.*

When there has been unusual difficulty in the extraction of the lens, entailing much instrumental interference, an ice compress is applied immediately after the operation, and is kept on for the next three days, or until the risk of corneal suppuration is past.

When the lens has been extracted through a corneal wound without iridectomy (as in the Le Brun operation) eserine is dropped into the eye twice daily for the first two days or until the wound is healed. Atropine is then used occasionally, so as to prevent iritis being set up by the contracted iris coming in contact with fragments of lenticular substance.

Secondary Operations.—After needling opaque membrane or iridotomy for closed pupil ice compress is applied for the first two days, atropine is dropped into the eye six or eight times daily, and iritis is treated by leeching, &c., as already described. The use of atropine should not be continued too long in such cases, as occasionally increased tension comes on under its use.

Iridectomy.

After the operation a cold wet compress is laid over the eye and removed about five hours later, when dry dressing is applied.

In glaucoma, where one eye only is affected, eserine is dropped into the other eye three or four times daily for a few days, so as to prevent the rise of tension which not unfrequently occurs when it is left to itself. This treatment is also advisable when one eye has been *excised* for glaucoma while the other eye remains sound.

* In the last 200 cases or so under my charge I have seen no case of failure of operation due to this cause. This good result I mainly attribute to the great care taken by Mr. Burnham (then Assistant House-Surgeon) of the instruments under his charge.

ON THE CONDITION OF THE OPTIC NERVE, CILIARY BODY, AND IRIS, IN INCREASED TENSION.

By W. A. Brailey, M.D., and Walter Edmunds, M.D.

In every case of increased tension, whether accompanied by retinal hæmorrhage or not, and whether primary, or secondary to injury, corneal ulcer, or inflammation of the uveal tract, there is found some change in the optic nerve. This in the majority of cases examined presents itself as an excessive amount of a tissue roughly resembling, though far more dense, than the ordinary fibrous stroma of the nerve.

The nerve in transverse section exhibits numerous apertures of blood-vessels, each surrounded by concentrically disposed fibrous tissue, in which is some excess of staining nuclei.

The zones thus constituted seem at first sight isolated from each other, but a closer examination shows that they are connected by thinner fibrous bands. The whole doubtless is merely the fibrous stroma of the nerve hypertrophied especially in the neighbourhood of its vessels. These are larger and far more numerous than usual, and generally show some proliferation of their endothelium. The nerve-tissue proper shows some increase of its own nuclei, and its bundles present in section very irregular shapes, owing to the encroachment on them of the connective tissue.

In the cases recited in the Curator's Report this condition is indicated by the term *Vascular Sclerosis* of the optic nerve.

In others the fibrous zones are even thicker than these, and contain in their centre only the small lumen of a blood-vessel, or even present no indication that they have ever contained one. They press upon, and leave little space for, the proper nerve-tissue. The innermost nerve-sheath is thickened, and the whole diameter reduced.

To this condition, which appears from a comparison of the different cases to be a more advanced or a more severe form than the previous, we shall apply the term *Sclerosis*.

But a certain number present nothing more than a swollen optic disc accompanied with neuritis, by which term we imply an increase of the deeply-staining nuclear bodies of the fibrous stroma of the nerve.

These are probably due chiefly to a multiplication of the connective tissue corpuscles normally existent there. It is possible, however, that some may be nuclei in the walls of newly-formed blood-vessels, for fine focussing gives occasional indications of a parallel lineal arrangement. Some may also be migrated leucocytes.

Concurrently with this neuritis there is found an inflammation in the space between the inner and outermost sheaths of the nerve. The loose fibres which are found here normally become swollen, and covered with newly-formed staining nuclei.

There is still another condition frequently seen, in which less-defined zones of translucent substance containing some nuclei, occupy the place of the dense fibrous tissue above described in vascular sclerosis.

The central artery of the nerve is also the subject of morbid changes. Its proper walls are seen to shade off into the dense fibrous tissue, which occupies the position of its sheath, and the looser tissue constituting its sheath-space, and to blend externally with the hypertrophied stroma of the nerve.

Sometimes, however, as in very early cases of increased tension, we see nothing but an increase of the vasa vasorum in size and number. This is probably the first stage, from which the others are transitions.

Sometimes, again, the sheath is densely packed with nuclear bodies, probably leucocytes escaped from the vasa vasorum in consequence of their condition. The proper wall of the artery is also thickened by inflammation.

In some cases a translucent substance, similar to that

previously described as surrounding the newly-formed blood-vessels of the septa, fills the sheath-space of the central vessels: it gradually becomes fibrillated, loses the few nuclei it possesses, and finally constitutes the dense connective tissue in which the thickened artery is imbedded, and by which it is compressed.

From a comparison of the histories of the cases, it would appear that neuritis with a distended vascular sheath constitutes an earlier stage than that with translucent exudation, that this fibrillates into the vascular sclerosis stage, till finally sclerosis of the nerve, with a corresponding condition of the walls of the central artery, is attained. These changes are passed through with greater rapidity and certainty according as the age of the patient happens to be advanced.

Sclerosis is especially seen in hæmorrhagic glaucomas, as they are always severe forms of the disease. Though Poncet described in 1873 a case of hæmorrhagic glaucoma in which he observed sclerosis of the nerve, little attention has been directed to the condition of the deeper parts of the nerve in this affection.

Buphthalmos exhibits in its nerve the same appearances as in the other forms of increased tension in young persons. These are best exemplified in the glaucoma secondary to perforating corneal ulcers.

We have no reason to believe that sclerosis can occur even as a senile change without preceding inflammation. If this be slight (as it appears to be in most cases of primary glaucoma) the disc will give no decided ophthalmoscopic indications as to the condition of the optic nerve. In the cases where we have found it well marked, as after excision for perforating corneal ulcers and injuries, the disc has rarely been visible with the ophthalmoscope.

It will hereafter be discussed as to whether these conditions of neuritis and sclerosis occur previous or subsequent to the increased tension. We may state here, however, that we are strongly of opinion that in primary glaucoma there is a pre-glaucomatous morbid change in the nerve. In the

cases of corneal ulcers and wounds we have evidence that the neuritis closely follows upon the corneal inflammation, and precedes the increased tension.

The ciliary body is also the subject of a similar morbid condition, though not with so great an uniformity of result as the optic nerve.

It may be added that the general conclusions arrived at here are in agreement with those of previous communications by one of the authors as to atrophy of the ciliary muscle (R.L.O.H. Reports, vol. ix, part 2), and the presence of enlarged blood-vessels (vol. ix, part 3). With regard to the latter condition, a stricter test for enlargement is here imposed, those cases only being thus recognized the calibre of whose arteries attains to at least twice the average normal diameter. In both papers the inflammation of the ciliary muscle was mentioned; its sclerosis, however, passed unnoticed.

In primary glaucoma the conditions most usually seen at excision are atrophy of the muscular (especially the circular) bundles, abundant dense fibrous tissue between their scanty remains, a fibrous condition of the ciliary folds, and in these either dilated and varicose capillaries, or a total absence of blood-vessels. The whole thickness of the ciliary body is reduced, it may be to even less than a third of its natural size. The iris is atrophied, more especially near its periphery. When the atrophy of the ciliary body is extreme, the pigment cells on its internal surface may be scattered and destroyed.

The longitudinal fibres of the muscle are also affected; their origin from the neighbourhood of the sclero-corneal junction instead of being of considerable thickness from without inwards is reduced to a pointed process. The diminution in size and alteration in shape of the ciliary body, due to its sclerosis and atrophy, approximate its internal surface, and more especially its apex, to the origin of the ciliary muscle, and in this movement the ciliary body necessarily carries with it the peripheral attachment of the iris.* The apex of the ciliary body is at the same time drawn backwards

* See page 93 and Curator's Report, Plate III.

towards the insertion of the muscle, which is the other fixed point.

The result of these two movements is the removal of the apex of the ciliary body to a position behind and external to that which it previously occupied, and consequently to make tense the fibres of the zonula. This agrees with what Roeder observed during life as to the extreme tension of the suspensory apparatus of the lens in glaucoma.*

Another result is that the angle of the anterior chamber of the aqueous, instead of being a little in advance of the origin of the longitudinal muscular fibres, is pulled back much posterior to it. Further, the angle instead of being rounded and open is reduced to a mere chink.

The ciliary body presents sometimes an inflamed condition, which is especially severe in the cases of glaucoma shortly following or complicated by corneal ulceration. In primary glaucoma, however, the inflammation is usually less acute. The ciliary body and iris present numerous nuclear bodies, scattered uniformly throughout their connective stroma, and especially in the immediate neighbourhood of, or even in, the muscular fibres themselves. This inflammation is, as a rule, more conspicuous in cases where the ciliary body is comparatively but little diminished in size, and in our opinion it is the cause of the subsequent atrophy.

Between inflammation and atrophy there appears to be an intermediate stage which consists in the conspicuous presence between the nucleated muscular bundles of a translucent material similar to that above described as occurring in the optic nerve.

Some cases in our possession exhibit the inflammation far more pronounced than usual. In some of them it is accompanied by much episcleritis. One case gives colour to the supposition that inflammation may travel along the track of the anterior ciliary arteries till it reaches and implicates the ciliary body. (See Curator's Report, Plate II, fig. 6.)

* Roeder, Ueber die gemeinschaftlichen Ursachen von Glaucom, &c., 1879.

This inflammation we believe to be the first stage of disease in the muscle, and, as in the case of the optic nerve, we are strongly of opinion that this condition precedes the establishment of increased tension.

As to the enlarged vessels of the ciliary region they may be found in company with any stage, but they are most conspicuous and most uniformly present in those cases in which glaucoma follows quickly on an accident (see Group III, p. 114). They certainly bear no general relation to the amount of inflammatory disturbance. The thinning of their walls is far greater than can be accounted for by their distension. They occasionally give indications of a varicose dilatation. Sometimes the arteries are not equally affected, and the same remark applies to the changes which are found in the ciliary body. They sometimes are confined to one part, the other part of the circle being unaffected.

This shows that the changes are not the result of increased tension, which would of course act equally on all parts of the circle of the ciliary body.

In cases of corneal perforation where the lens is thrust forward against the cornea, the much stretched and cystic anterior part of the retina is sometimes found lying on the internal surface of the ciliary body.

The iris has been described as nearly brought into contact with the cornea by the sclerosis and advanced atrophy of the ciliary body. The same effect can be produced by atrophy alone: it can also be produced in cases where the ciliary part of the sclerotic has yielded, the sclero-corneal junction retaining its position. In these last, however, it is rather a consequence than a cause of increased tension.

In certain cases, however, a somewhat different condition is found. In these the ciliary body is only atrophied to a partial extent, sufficient to draw back the base of the iris behind the level of the canal of Schlemm, but not sufficient entirely to destroy the rounded shape of the angle of the anterior chamber. In these cases of glaucoma the iris is still found to be applied, though not at its extreme base (which is sepa-

rated from the anterior wall of the chamber by the rounded angle), but at that part which is opposite to the canal of Schlemm.

That the iris should be applied just in this position, and neither in front nor behind it looks, decidedly, as if there were some suction of this part of the iris towards the open meshwork of the ligamentum pectinatum. The appearances in many other specimens support this supposition. But in most cases fluid is not thus entrapped in the angle of the chamber, but the iris periphery is applied throughout.

After a corneal ulcer has perforated, the iris is usually applied to the whole posterior surface of the cornea, through the application is sometimes limited to the sector corresponding to the ulcer and to the peripheral part of the iris in the remainder of its circumference.

In increased tension consequent on serous inflammation of the uveal tract the ciliary muscle and iris do not become atrophied nor is there any sclerosis. There is considerable inflammation of both ciliary body and iris, and there are also staining corpuscles in considerable numbers on the posterior corneal surface, in the ligamentum pectinatum, and round the canal of Schlemm. The choroid is inflamed and shows some evidence of increased fluid in the perichoroidal space. The nerve presents neuritis, but no sclerosis. The iris periphery is not applied, but indeed is rather widely separated from the cornea.

Sclerosis of ciliary body is most common in hæmorrhagic glaucoma, being present in 85 per cent. of the cases.

Then follow primary glaucomas without hæmorrhage with 58 per cent., and after these the cases where glaucoma has followed accidents with 71 per cent. These last cases, however, are on the average of much less duration than the others.

In glaucoma, secondary to perforating corneal ulcers under 20 years of age, the degree of sclerosis is never very great, and it is present even moderately in only 20 per cent. of the cases.

But when persons over 20 years of age are from the same

cause the subjects of secondary glaucoma, sclerosis is found greatly developed in 25 per cent., and moderately in 25 per cent. of the cases.

Sclerosis is never seen without some atrophy, far more often the amount is considerable. Atrophy, on the other hand, is often fully developed in young subjects without any sclerosis.

We believe this sclerosed condition is the cause of the cataract occurring in glaucoma, and that a similar but slight degree of this condition may be the cause of ordinary senile cataract through mal-nutrition of the lens.

The changes in the ciliary body then are strictly comparable with those in the optic nerve. Though in young subjects there is this difference, that while inflammation of the muscle usually leads to atrophy alone, in the nerve it usually ends in sclerosis. In old persons sclerosis is the goal of both conditions.

Since the above was written more extended and exact microscopical measurements have shown that the closure of the angle of the anterior chamber of the aqueous is principally due to a contraction of the fibrous band known commonly as the ligamentum pectinatum. The contraction of the innermost layer of this draws the connective tissue framework of the iris, which has its origin here, towards the termination of the posterior elastic lamina of the cornea. That of its outermost layer draws together to a point the meridional fibres of origin of the ciliary muscle, and contracts, through its connexion with their connective tissue stroma, the ciliary processes.

The ligamentum pectinatum may also alter in position, as before explained, by the drawing back of its posterior angle due to the development of connective tissue in the ciliary body generally.

CURATOR'S PATHOLOGICAL REPORT ON

By W. A.

Short Abstracts of the History

THE following cases of Glaucoma have been divided into groups, the members the most recent:—

The Pathological Reports deal especially with the parts which are most *Iris, Canal of Schlemm, Optic Disc, Optic Nerve,* and *Retina.* Other changes special interest in each case are called attention to under the head of *Remarks.* used, is explained in the paper immediately preceding this. The term *Hyaline* highly refracting, containing very few or more generally no nuclei. Capil- open on transverse section. *Translucent* implies an ill-defined, almost struc- means that their walls are thicker than normal, and contain an excess of Optic Nerve. P. C. R. = Pars Ciliaris Retinæ. C. of Sch. = Canal of

GROUP 1.—*Twenty-nine cases of Primary Glaucoma,*

History.	Ciliary muscle.	Iris.	Canal of Schlemm.
No. 354. Wm. R., æt. 43. He attended Mr. Hulke as out-patient 15 months ago. Then the diagnosis was anæmia of o.d. of *Left* eye. He had gout at that time. Within the last 1½ years he has had several fits, apparently epileptic. Seven weeks ago he came again with his left eye bad. It had superficial ulceration of cornea and hypopyon and T. 2. Paracentesis was done 4 weeks before excision.	Of good size and shape, much inflamed. Some sclerosis of tissue between circular fibres.	Inflamed periphery applied to cornea; lower limb involved in a corneal ulcer.	Access closed by iris.
No. 395. John D., æt. 46. Two months ago he attended Mr. Adams as out-patient with embolism of art. cent. ret. of *L.*, and direct aortic murmur. V = $\frac{20}{200}$ then. It had been defective 1 month. He had been drinking much of late. He comes now with T. 3, deep a.c. and no p.l. Pupillary margin irregular, with small posterior synechiæ. Has had severe pain 1 month.	A little inflamed; moderately atrophied; thickness = 0·46 mm.; slight sclerosis of tissue between circular fibres.	Periphery separated by only a narrow chink from cornea to which it has evidently been applied. At other points of ciliary circle the periphery is just adherent.	Lig. pect. and c. of Sch. somewhat inflamed.

THE MORBID ANATOMY OF GLAUCOMA.

Brailey, M.D.

and Pathology of Hospital Cases.

of which are arranged in the order of duration of the disease, beginning with

commonly the subjects of the greatest morbid change, *e.g.*, the *Ciliary Muscle*, are indicated under the head of *Other Pathological Conditions.* Any points of The sense in which the terms *Neuritis*, *Vascular Sclerosis*, and *Sclerosis* are is applied to blood-vessels when their walls are thick, glassy-looking, and laries of this description are usually larger than normal, and stand widely tureless semi-transparent substance. *Hypertrophied*, as applied to arteries, nuclear bodies. A. C. = Anterior Chamber. O. D. = Optic Disc. O. N. = Schlemm. P. L. = Perception of Light.

uncomplicated by Retinal Hæmorrhages.

Blood-vessels of ciliary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
Not enlarged.	Shallow central pit. Margins swollen.	Moderate vascular sclerosis.	Distended with red corpuscles. Sclerosis of adventitia of arteries of papilla and obliteration of what has been described as the lymph-space.	Considerable episcleritis.	Previous white atrophy of disc. *See History.*
Moderately enlarged.	Shallow central pit.	Much neuritis.	Walls hypertrophied and containing excess of nuclei.	A little episcleritis. The vitreous contains some small blood spots apparently from cil. processes below.	Previous cardiac disease and embolism. *See History.* (?) Glaucoma hæmorrhagicum.

GROUP 1.—*Twenty-nine cases of Primary Glaucoma.*

History.	Ciliary muscle.	Iris.	Canal of Schlemm.
No. 401. Amelia C., æt. 69. 3 months ago while in bed with pneumonia an acute attack of glaucoma of the *Left* came on with severe neuralgia of teeth of left upper jaw. T. 2.	Very thin, much structureless substance between bundles of muscle fibres now just fibrillating.	Periphery adherent.	Access closed by iris.
No. 204. Wm. B., æt. 60. Cataract extraction 3 yrs. ago, followed by some iritis. Eye needled 3 months later. Vision fair till 3 months ago, when glaucoma set in. Iridectomy 2 mons. ago, but T. not relieved. Iridectomy again 2 weeks ago in opposite direction, followed by panophthalmitis.	Slight atrophy; 6 mm. Some sclerosis of antero-internal part of muscle. Great numbers of pus-corpuscles on ciliary body and in vitreous.	Completely removed by iridectomy. Abundant pus in ant. chamber.	Lig. pect. somewhat inflamed, possibly preventing access.
No. 241. Jane W., æt. 55. Glaucoma of *Left* 5 mons., beginning with neuralgia of frontal region and bridge of nose; photophobia and lacrimation. At excision T. 2. Lens dislocated; upper edge in a.c. just within pupillary margin. History of a blow some time previous to the failure of sight.	Great atrophy and sclerosis as above.	Atrophied; periphery adherent to cornea.	Access closed by iris.
No. 88. Woman, æt. 60. Glaucoma of about 5 months' duration. History very defective.	Extreme atrophy; translucent stage of sclerosis.	Periphery adherent.	Access closed ..
No. 763. John James, æt. 54. Eye dim 5 months. Both sugar and albumen were found in his urine 4 months ago. Had noticed when his eye was first dim that he passed much water. Shortly before excision albumen only found, and in small amount. At excision neither albumen nor sugar.	Some atrophy and inflammation; translucent stage between muscular fibres and in ciliary processes.	Periphery adherent.	Access closed ..

uncomplicated by Retinal Hæmorrhages.

Blood-vessels of ciliary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
Much enlarged; walls of arteries extremely thin.	Moderately deep cup, extending only over half its area.	Neuritis and increased blood-vessels; structureless substance filling their sheaths.	Specimens not good.	Sclerosis of blood-vessels of choroid, especially of adventitia.	.. ..
Slightly enlarged. Walls thick.	Swollen. Blood-vessels of papilla have their walls a little sclerosed.	Moderate vascular sclerosis, more advanced than in papilla.	Some have hypertrophied walls. Others have their positions occupied by a round mass of nuclei which fill the lumen.	Increase of nuclei in nerve-fibre layer of retina, and on inner surface of ciliary body. Choroid containing some diffused increase of nuclei, and is somewhat œdematous.	Diffuse small hæmorrhages in retina. Much episcleritis.
Not enlarged.	Centre of lam. crib. pushed back, its concavity filled by nuclear tissue.	Sclerosis. Strong bands are seen running longitudinally.	Walls hypertrophied.	Choroid has slight occasional patches of inflammation, somewhat like plastic.	.. ..
Not enlarged.	Central pit, lam. crib. pushed back into a curve.	Neuritis, translucent substance in perivascular spaces.	(?)	.. ..	Specimen not from Moorfields Hospital.
Enlarged ..	Very shallow cup, occupying $\frac{2}{3}$rds. of o.d. Lam. crib. sclerosed.	Neuritis, translucent substance round vessels. Lam. crib. thick and translucent looking.	Walls of smaller retinal arteries hyaline and thick.	Slight episcleritis.	Albumen, but no retinal hæmorrhages. Apparently no cardiac disease. Urine not examined microscopically.

GROUP 1.—*Twenty-nine cases of Primary Glaucoma,*

History.	Ciliary muscle.	Iris.	Canal of Schlemm.
No. 151. Ann J., æt. 55. She attended Mr. Streatfeild as out-patient 13 years ago, then diagnosis was old choroido-retinitis; she read J. 2 with + $\frac{1}{12}$. Ten months ago acute neuralgia of *R.* eye and whole *R.* side of head came on. The eye was much inflamed. P.l. soon went. In 7 weeks inflammation of eye subsided. Pain continued, though less severe, till excision.	Moderate atrophy, good shape, abundant sclerosis.	Periphery adherent.	Access closed ..
No. 406. Henry T., æt. 49. Glaucoma of *L.* 11 months. T. +. Staphyloma at inner side of o.d. Choroiditis disseminata at macula. *R.* myopia = 13 D.	Fair size, moderate sclerosis of circular fibres.	Periphery widely free from cornea.	Open .. ..
No. 144. John D., æt. 58. Glaucoma for 1 year. Painful 9 months ago. Cataract and no p.l. since 6 months.	Atrophied; inflamed.	Periphery adherent.	Access closed ..
No. 441. George S., æt. 73. *R.* failing slightly 2 years. Severe neuralgia of *R.* brow 8 weeks ago. P.l. ceased then.	Atrophied; sclerosis of muscle and cil. processes.	Periphery adherent.	Access closed ..
No. 466. Joseph B., æt. 68. Eye has failed since 2 years. The cataractous lens was removed from it 13 months ago while the T. was still in excess. Iridectomy was done upwards then. Tension soon recurred, and continued till excision, bulging the iris forwards.	Inflammation and moderate atrophy.	Periphery adherent.	Access closed below by iris, above by inflammatory exudation.
No. 74. Margaret H., æt. 71. Eye faded gradually without pain 3 years ago till no p.l. Severe pain 2 years ago, relieved by iridectomy. Pain recurred 1 year later, and continued more or less till excision.	Much atrophy; some inflammation; much translucent substance between fibres.	Periphery adherent, but limb gone at upper part of cil. circle.	Access closed ..

uncomplicated by Retinal Hæmorrhages.

Blood-vessels of ciliary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
Enlarged ..	Shallow universal cup. Lam. crib. sclerosed.	Sclerosis in bands. Slight neuritis between them.	Some have their walls hypertrophied. Others are mere fibrous cords. Many have much irregular black pigment in their sheaths, and perhaps in their lumen.	In some places the ch. and ret. adhere, and the pigment epithelium is scattered and destroyed. There is an accumulation in retina (q.v.).	.. ..
Not enlarged.	Characteristic of myopia. Not cupped.	Vascular sclerosis, ill-defined.	Walls hypertrophied.	Adhesion in various places between ch. and ret. obviously from old ch. diss.	Extra deep a.c.
Enlarged ..	Deep cup. ..	Neuritis ..	Walls a little hypertrophied.	A little general increase of nuclei in choroid.	A.c. fairly deep at centre.
Enlarged ..	Pit-like cup, not up to margin.	Neuritis and vascular sclerosis.	Apparently normal.	.. ..	.. ..
Not enlarged.	Pit not up to margin.	Neuritis ..	No specimen ..	Some nuclei in sheaths of arteries of papilla.	.. ..
Not enlarged.	Shallow cup. Lam. crib. sclerosed.	Neuritis, faintly fibrillated translucent substance surrounding blood-vessels.	No specimen ..	.. ..	.. ..

GROUP 1.—*Twenty-nine cases of Primary Glaucoma,*

History.	Ciliary muscle.	Iris.	Canal of Schlemm.	
No. 35. John C., æt. 44. *R.* faded gradually and without pain 3½ years ago. No p.l. one year later. *L.* failed in same way 1½ years ago, but became painful later. Iridectomy in *R.* 4 months before excision.	Atrophied, inflamed in translucent stage of sclerosis.	Inflamed somewhat; periphery adherent.	Access closed	..
No. 746. David S., æt. 39. *L.* faded painlessly after slight accident 15 years ago; last saw light 2½ years ago. Pained him first 3½ years ago.	Atrophied, inflamed, early stage of sclerosis.	Periphery adherent.	Access closed	..
No. 474. John S., æt. 68. Glaucoma subacute 4 years, recent pain. Soft leucoma of cornea from ulcer, apparently more recent.	Extreme atrophy; much translucent substance between fibres and in ciliary folds.	Much atrophied; periphery adherent; apex entangled in leucoma of cornea.	Access closed	..
No. 368. Naomi E., æt. 65. Sight of *L.* began to fail 4 years ago with pain and lacrimation. No p.l. since 1½ years. Severe pain lately. Soft leucoma of cornea, apparently recent. *R.* glaucomatous 2½ years. Now T. 2 and posterior synechiæ.	Atrophy; much sclerosis.	Periphery adherent; lower limb in leucoma.	Access closed	..
No. 64. Eleanor B., æt. 49. Bad off and on 8 years. Severe symptoms 2 years. No p.l. for 1 year.	Structure much atrophied and inflamed. Some translucent substance between its fibres.	Inflamed; periphery adherent.	Access closed	..
No. 178. Stephen G., æt. 41. *R.* failed quickly 10 years ago. No pain till 5 weeks ago. Attempted iridectomy 3 weeks later.	Fair size and shape; much inflammation.	Inflamed, periphery adherent. Uveal pigment rolled into view by the contraction of some inflammatory material on its front surface.	Access closed	..

uncomplicated by Retinal Hæmorrhages.

Blood-vessels of ciliary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
Not enlarged.	Deep cup, with excavated sides.	Neuritis, with new blood-vessels. It is in the translucent stage of sclerosis.	Neither hypertrophied nor hyaline.	.. ..	.. ..
Enlarged ..	Deep cup ..	Neuritis, translucent substance round blood-vessels.	Neither hypertrophied nor hyaline.	.. ..	.. ..
A little enlarged.	Very shallow cup.	Neuritis, much translucent substance around blood-vessels.	No specimen ..	Sclerosis of sheaths of blood-vessels of choroid.	.. ..
Not enlarged.	Shallow cup.	Vascular sclerosis, a much earlier stage than in muscle.	No specimen ..	.. ..	A much more advanced stage of sclerosis in muscle than in nerve.
Enlarged ,,	Moderately deep cup.	Much neuritis, nuclei in perivascular space, and some translucent substance.	Arteries have rather hypertrophied walls. Capillaries have walls hyaline and gaping.	.. ..	.. ..
Enlarged, full of blood corpuscles.	Deep cup. Abundant translucent substance in sheath of central artery.	Much neuritis, very early stage of sclerosis.	Neither hypertrophied nor hyaline.	.. ..	.. ..

GROUP 1.—*Twenty-nine cases of Primary Glaucoma,*

History.	Ciliary muscle.	Iris.	Canal of Schlemm.
No. 376A. Sarah H., æt. 70. Old glaucoma. T. 2. Iridectomy done 8 years ago with some benefit, but T. recurred after 1 year.	Much atrophy and much sclerosis.	Periphery adherent to scar of iridectomy and to cornea.	Access closed ..
No. 353. Sarah H., æt. 68. Subacute glaucoma many years. Attempted iridectomy upwards 10 days before excision. Traumatic cataract. Bulging cicatrix with lymph, &c.	Moderate, recent inflammation. Sclerosis. Inflammatory exudation on internal surface of ciliary body.	Periphery adherent, inflamed.	Access closed ..
No. 340. Woman, æt. 56. Glaucoma of doubtful duration. Iridectomy a few weeks before excision. Sympathetic inflammation of uveal tract of other eye.	Fair, sclerosed. Nuclei on internal surface of ciliary body.	Thick, with inflammatory new formation in scar of iridectomy; periphery applied.	Access closed ..
No. 197. Solomon S., æt. 54. Old glaucoma of *L.* It has been painful for 2 years. Has had iridectomy in three different directions without relief of T. At excision T. 2.	Very much atrophied, moderate sclerosis.	Atrophied limb; removed from some sections by the operation.	Access closed by adherent base of iris.
No. 86. Mary B., æt. 63. *L.* faded 30 years ago. No p.l. in 1 year. Recent pain and inflammation, with onyx and hypopyon. At excision T. +.	Complete atrophy; much inflamed.	Periphery adherent, atrophied, much inflamed.	Access closed by adherent base of iris.
No. 67. Charles R., æt. 55. Chronic glaucoma of many years' duration.	Atrophied, inflamed; some translucent substance.	Periphery adherent, atrophied.	Access closed by adherent base of iris.

uncomplicated by Retinal Hæmorrhages.

Blood-vessels of ciliary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
Enlarged ..	Shallow cup.	Marked sclerosis.	(?)	Optic nerve shrunk to half its proper size. Sclerosis of outer walls of blood-vessels of choroid.	.. ..
Not enlarged.	Bad specimen	Marked sclerosis.	Neither hypertrophied nor hyaline.		.. ..
Not enlarged.	No specimen.	Marked vascular sclerosis. Dense connective tissue surrounding central artery and vein.	No specimen ..	Inflammation of choroid, plastic in character.	The nerve is shown in transverse section, and is more densely sclerosed than the muscle. Not from Moorfields Hospital.
Enlarged ..	Deep cup ..	Vascular sclerosis.	Some have their walls thickened and hyaline.	.. ..	.. ..
Not enlarged.	Shallow cup.	Much neuritis.	Large tracts where choroid and retina atrophied, and adherent. Walls of blood-vessels neither hypertrophied nor hyaline.	.. ..	.. ..
Enlarged ..	Much nuclear tissue in front of concave lam. crib.	Very much neuritis, some ill-defined translucent substance.	Walls of blood-vessels as above.	Long ciliary arteries are large.	.. ..

GROUP 1.—*Twenty-nine cases of Primary Glaucoma,*

History.	Ciliary muscle.	Iris.	Canal of Schlemm.
No. 5. Elizabeth B., æt. 56. Glaucoma of many years' duration. Pupillary margin irregular, with posterior synechiæ. Cornea is leucomatous at one place.	Atrophied, much inflamed; much early sclerosis.	Periphery adherent.	Access closed by adherent base of iris.
No. 265. Man, æt. 72. *R.* cataractous 20 years, but glaucomatous only 6 months apparently. Now T. 3.– Pain.	Moderately atrophied; much sclerosis.	Periphery free. It has apparently never been applied.	Open .. ..

The following two cases must be added, agreeing as they do in all important of their symptoms and morbid appearances, which would cause them probably

No. 452. Rebecca B., widow, æt. 56. History imperfect on account of the insanity of the patient. There is much iritis and episcleritis. The aqueous is turbid, and the a.c. in its centre is of fair depth. T. 2.	Atrophied and much inflamed.	Inflamed, atrophied, periphery adherent.	Access closed; lig. pect. and c. of S. much inflamed.
No. 492. Henry C., æt. 19. *R.* faded gradually 9 years ago; inflamed 2½ years. At excision T. 2. A.c. of good depth.	Atrophied and much inflamed.	Inflamed, atrophied, periphery adherent.	Access closed; lig. pect. inflamed.

uncomplicated by Retinal Hæmorrhages.

Blood-vessels of ciliary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
Enlarged somewhat.	Deep cup ..	Great neuritis; some sclerosis of one side of nerve in especial.	Walls of blood-vessels as above.	.. ..	.. ..
Not enlarged.	No specimen.	Vascular sclerosis, pushing aside nerve bundles somewhat irregularly.	Walls of blood-vessels as above.	.. ..	Not excised in Moorfields Hospital. Transverse sections of O.N.

pathological points. They differ only in the more inflammatory character to be ranked clinically under the head of Inflammatory Glaucoma.

Not enlarged.	Shallow universal cup.	Neuritis and commencing vascular sclerosis.	Walls of arteries hypertrophied and dissociated.	Cells of pigment epithelium of retina destroyed. Pigment increased, black and scattered; outer granules and rods and cones gone. Much episcleritis following wall of anterior ciliary artery to muscle.	Patient, a stout healthy-looking woman, has been in asylum 11 years, on account of dementia. No history of syphilis known.
Not enlarged.	Deep cup ..	Neuritis and commencing vascular sclerosis.	Retinal arteries are represented by strong fibrous cords, mostly impervious.	Pigment epithelium as above. Episcleritis.	No history of syphilis or any serious illness.

GROUP 2.—*Twenty-one cases of Glaucoma,*

History.	Ciliary muscle.	Iris.	Canal of Schlemm.
No. 483. Edward E., æt. 46. *R.* failing since 6 months. Three months before excision attended Mr. Tay, whose diagnosis was *R.* Retinitis with hæmorrhages. V. = shadows. Numerous hæmorrhages near macula and small white spots also. *L.* V = $\frac{2}{20}$. Has had gout lately. *At excision* T. 3. Ac. good.	Atrophied, thickness = ·36 mm., much inflamed.	Inflamed, periphery adherent.	Access closed ..
No. 777. Amelia D., æt. 50. *R.* old blind eye. Blow 6 weeks ago. Since then painful. Glaucoma probably of longer duration than 6 weeks, judging from the optic disc. History more fully given in vol. ix, p. 398.	*Upper part*, much atrophy, some inflammation, abundant translucent material just fibrillating. *Lower part*, less atrophy and inflammation, otherwise as above.	*Upper part*, periphery adherent. *Lower part*, periphery just adherent.	Access closed ..
No. 177. Elizabeth R., æt. 79. Glaucoma of *R.* of 8 or 10 wks.' duration. *L.* normal. History more fully reported in vol. ix, p. 394.	Of fair size, inflamed, much translucent material near centre of muscle.	Of good size, separated by narrow chink from cornea.	Access probably closed.
No. 364. Joseph R., æt. 40. *L.* eye quite well till 9 weeks ago, then shooting pain. Came here 3 weeks ago. Then bulging of lower part of cornea, upper part tilted upwards and pretty transparent. History more fully in vol. ix, p. 419. It is now thought that the corneal change is secondary to the glaucoma.	*Upper part*, mod. atrophy and sclerosis of tissue between muscular bundles. *Lower part*, much atrophy and sclerosis.	*Upper part*, periphery just adherent. *Lower part*, atrophied, and much adherent to the bulged cornea.	Access closed ..

accompanied by Retinal Hæmorrhages.

Blood-vessels of ciliary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
Not enlarged.	Shallow central pit; translucent substance in sheath of art. centralis in nerve.	Neuritis, translucent substance round numerous small blood-vessels far back.	Walls hypertrophied.	Hæmorrhages numerous, and mostly capillary in intergranular layer.	No albumen in urine. No cardiac bruit. Had an appearance as if of excess in alcohol.
Upper part somewhat enlarged. *Lower part* of normal size.	Cupped ..	Neuritis, abundant translucent material within lymph-sheaths of blood-vessels near papilla.	Arteries have hyaline walls.	.. ..	Changes in the upper ciliary region much more advanced than in lower.
Somewhat enlarged.	Shallow depression of disc.	Neuritis especially in front, marked vascular sclerosis. Sclerosis of adventitia of cent. artery.	No specimen ..	Choroid healthy, but its epithelium, as is not unusual, in a colloidal condition.	Changes more advanced in optic nerve than in cil. muscle.
Not enlarged.	Shallow pit..	Vascular sclerosis.	Walls hypertrophied.	Hæmorrhage most in intergranular layer.	Some yielding of the ciliary portion of the sclerotic above; the region of sclero-corneal junction has not yielded.

GROUP 2.—*Twenty-one cases of Glaucoma,*

History.	Ciliary muscle.	Iris.	Canal of Schlemm.	
No. 122. Sarah P., æt. 53. *L.* watered 9 months ago. Came dim 3 months later. Came here 4 months ago; then albuminuria; + T. and severe pain. Hæmorrhage in retina. Iridectomy upwards, followed in 3 days by iridectomy downwards. Pain relieved for 3 weeks. Then recurred, and sclerotomy done with temporary relief.	Moderate atrophy, inflamed.	Inflamed, periphery adherent.	Access closed	..
No. 61. Eliza B., æt. 25. *L.* glaucomatous 9 months at *excision*. Eye enlarged. Numerous ciliary staphylomata. Abundant dark blood between ch. and det. ret. A.c. of good depth. This case is mentioned in vol. ix, p. 410.	Structure thin and inflamed.	Inflamed, periphery adherent.	Access closed	..
No. 49. Stephen H., æt. 73. *L.* faded imperceptibly 9 months ago; pain came 2 months ago. Radial artery atheromatous. Pulse slow.	Atrophied and sclerosed (translucent stage).	Inflamed, periphery adherent.	Access closed	..
No. 160. Jane R., æt. 38. *L.* came bad 12 months ago. Severe pain for last 4 months. At excision T. 3. Large staphyloma involving a large extent at upper and outer part of equator. This case is mentioned in vol. ix, p. 410.	Atrophy, inflammation, and very early sclerosis.	Of fair size, base adherent.	Access closed	..
No. 270. Mary S., æt. 59. Pain in *R.* 14 months ago; lost p.l. in 4 months. Noticed white speck on cornea 4 months only. Severe pain for last 4 months. *L.* a little watery, but otherwise not affected.	Thin sclerosis of circular fibres: somewhat inflamed.	Inflamed periphery adherent.	Access closed	..
No. 143. Sarah D., æt. 78. Attended here 5½ years ago for an accident followed by leucoma of cornea of *R.* There appears to have been no tension till 18 months ago, when it came bad with pain and lacrimation. *L.* bad 6 months; pain 3 months; now T. 1.	Inflammation of radial fibres, especially circular atrophied, with abundant early sclerosis.	Periphery adherent.	Access closed	..

accompanied by Retinal Hæmorrhages.

Blood-vessels of ciliary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
Not enlarged.	Shallow cup.	Neuritis ..	No specimen ..	.. ..	Albumen in urine. A.c. filled with blood after the sclerotomy, which was very slowly absorbed.
Not enlarged.	Lam. crib. pushed back, cavity filled by nuclear tissue.	Neuritis, with new blood-vessels and early sclerosis.	No specimen. Ret. detached	General diffused nuclear increase in choroid, especially near staphyloma. Some episcleritis.	Staphyloma of sclerotic in ciliary region.
Enlarged ..	Shallow cup.	Neuritis and some vascular sclerosis.	Walls of arteries are hyaline.	.. ..	Changes in muscle more advanced at upper than at lower part of cil. circle.
A little enlarged.	A deep pit ..	Neuritis and early vascular sclerosis.	?	Diffused increase of nuclei in thickened ch. near optic disc.	Staphyloma of sclerotic at upper ciliary region.
Enlarged, full of blood corpuscles.	No specimen.	No specimen.	Arteries hypertrophied.	Dense leucoma of lower and outer part of cornea, with anterior synechia. A.c. exists elsewhere.	The leucoma appears to be secondary in time to the glaucoma.
Enlarged ..	Deep cup ..	Neuritis, early stage of sclerosis, with scattered nuclei.	Walls of arteries hyaline.	Dense leucoma of cornea. A.c. fairly deep.	.. ..

GROUP 2.—*Twenty-one cases of Glaucoma,*

History.	Ciliary muscle.	Iris.	Canal of Schlemm.
No. 289. Rebecca D., æt. 66. Glaucomatous attack in *L.* 2 years ago, with acute pain. *R.* T. 2 and pain. History more fully in vol. ix, p. 393.	Extreme atrophy and sclerosis.	Periphery adherent; iris sclerosed.	Access closed, and canal closed also.
No. 259. Mary S., æt. 74. *R.* faded quickly with pain 2 years ago. *L.* dim occasionally. Pupil sluggish. T. 1.	Extreme atrophy and sclerosis.	Sclerosed, periphery adherent.	Access closed ..
No. 786. Harriet G., æt. 62. *L.* glaucoma 2½ years. The sight went very quickly, with inflammation and pain. T. 2 now. *R.* somewhat defective, but T. n.	Atrophy and sclerosis.	Periphery adherent.	Access closed ..
No. 382. Eliza B., æt. 65. Attended here as out-patient 5 years ago, when the diagnosis was *Retinitis with hæmorrhages* in *L.*, and a trace of albumen was found in her urine. Six months later she had several slight attacks of hemiplegia, after each of which the V. was worse. Severe pain in *L.* 2 months. At excision of *L.*, systolic cardiac murmur. *R.* about normal.	Much atrophy and sclerosis of circular fibres and ciliary folds.	Periphery adherent.	Access closed ..
No. 425. Isabella D., æt. 64. *R.* Graefe extraction 8 years ago; fair result, but blow soon after discharge. Soon after this, T. 1. *At excision,* T. 3; cornea hazy. Iris bulged forwards; some blood in a.c.; pain. *L.* Graefe extraction 1 year before *R.*; did well; V. now good, but T. 1.	Atrophy, much sclerosis of muscle.	Sclerosis, periphery adherent, limb gone above.	Access closed ..
No. 167. George J., æt. 67. *L.* chronic glaucoma. No p.l. for 1 year. Iridectomy 5 weeks ago. Blood in a.c. first observed 3 days before excision. T. 3, severe pain lately. *R.* chronic glaucoma. Iridectomy 5 weeks ago. Now a.c. shallow. T. 1 (?)	Sclerosis, moderate atrophy.	Periphery adherent.	Access closed ..

accompanied by Retinal Hæmorrhages.

Blood-vessels of ciliary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
Enlarged, especially at lower part.	Moderate deep cup. Sclerosis of walls of blood-vessels of papilla.	Some neuritis and vascular sclerosis.	No specimen ..	Retina detached. Elongated thinned tract of sclerotic crossing inner equator. Anterior part of retina a little cystic.	.. ..
Not enlarged.	Shallow cup .	Much vascular sclerosis.	Walls moderately hypertrophied.	Retina detached.	.. ..
Enlarged ..	No specimen.	No specimen.	Varicose dilatations of capillaries of retina.	.. ..	.. ..
Enlarged ..	Shallow cup, sclerosis of arteries in papilla.	Vascular sclerosis.	Walls hypertrophied.	.. ..	Diagnosis 5 years before excision was *L.* Hæmorrhagic Retinitis, with a trace of albumen.
Not enlarged.	Cupped ..	Enormous degree of vascular sclerosis.	Walls of arteries and capillaries are hyaline.	Long internal ciliary artery is enlarged.	Old cataract extraction.
Enlarged ..	Slightly cupped.	Vascular sclerosis.	Hyaline thickening of walls of smaller arteries.	Abundant hæmorrhages in intergranular layers.	.. ..

GROUP 2.—*Twenty-one cases of Glaucoma,*

History.	Ciliary muscle.	Iris.	Canal of Schlemm.
No. 290. D. W., æt. 71. *R.* failed gradually 20 months ago. At excision some pain. T. 3; barest p.l. *L.* irritable.	Atrophy and sclerosis.	Periphery adherent.	Access closed, but canal of Schlemm patent.
No. 301. Sarah H., æt. 75. *R.* came bad 2 years ago while she was in bed with a broken leg. Pain then. At excision, T. 2. Dense leucoma of cornea; no anterior synechia. *L.* T. n.	Extreme atrophy. Only a few nuclei and a little sclerosis tissue remains. Ciliary folds much sclerosed.	Sclerosed, periphery adherent.	Access closed ..
No. 81. James D., æt. 35. *R.* failed gradually 3 years; much pain 2 years. *L.* T. n.	Inflamed, moderately atrophied.	Periphery adherent.	Access closed ..
No. 303. Wm. F., æt. 80. Glaucoma in *R.* about 12 months. Pain 6 weeks. *L.* also glaucomatous, but less severely so. Iridectomy in both 3 weeks ago. T. in *R.* not relieved. History more fully in vol. ix, p. 395.	Much atrophied, much sclerosis tissue in a rather early stage, and containing a few nuclei.	Limb removed at upper part by iridectomy; periphery free at lower part.	Access open, but some nuclei in lig. pect. and c. of S., which is very small.
No. 499. Amos B., æt. 48. *R.* came foggy 4 years ago. Now no p.l. for 3 years. Moderate pain 1½ years, and much inflammation and pain during the last 4 months. At excision T. 2. There is a staphyloma of the inner equator the size of a large pea. The a.c. is of fair depth, except at its margins, where it is obliterated. There are extensive posterior synechiæ. Lens cataractous and *in situ*.	Extremely atrophied, inflamed, early sclerosis.	Much inflamed; enlarged blood-vessels in its base.	Access closed ..

accompanied by Retinal Hæmorrhages.

Blood-vessels of ciliary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
Enlarged ..	Moderately deep cup, extending over the entire disc.	Neuritis, also sclerosis, especially of the sheath of the central artery.	Have hyaline walls.	.. ..	.. ..
Enlarged ..	Universal cup, not very deep.	Much vascular sclerosis.	A little hypertrophied. Capillaries gape; walls hyaline.	Some arteries of choroid much thickened in neighbourhood of optic disc. Anterior part of retina is very cystic.	Leucoma apparently secondary in time to the glaucoma.
Not enlarged.	Cupped ..	Much neuritis, some sclerosis, numerous nuclei in sheath of central artery.	Small blood-vessels of anterior part have walls much thickened and hyaline.	.. ..	.. ..
Not enlarged, but long internal ciliary arteries are vastly enlarged and thin-walled.	Shallow shelving cup.	Much vascular sclerosis.	Walls much thickened, especially those of the smaller arteries, which have their lumen nearly obliterated.	.. ..	No note as to condition of kidneys or heart.
Moderately enlarged.	Small cup ..	Neuritis and vascular sclerosis.	Walls of arteries hypertrophied, capillaries with aneurysmal dilatations.	Choroid at staphyloma inflamed, thin. Retina corresponding to it is very cystic.	The principal feature about the case is the marked inflammatory condition of the uveal tract and the cystic retina at the area of local staphyloma.

GROUP 3.—*Eight cases in which a blow or wound attended by comparatively slight which the eye has*

History.	Ciliary muscle.	Iris.	Canal of Schlemm.
No. 101. Hy. W., æt. 76. Minute perforating corneal wound 14 days ago. T. 1 at excision. In both eyes M = about 6 D. History more fully reported in vol. ix, p. 396.	Fair size and structure. Some translucent substance between bundles of circular fibres.	Periphery widely free, except at the one point, where prolapsed into internal orifice of the wound.	Wide open, except at the point where plugged by iris.
No. 371. Thomas M., æt, 40. Rupture of upper sclero-corneal junction by a blow 21 days before excision. T. 2. History more fully in vol. ix, p. 404.	Much atrophied, much sclerosis. Sclerosis and atrophy considerable, but not so great at lower part of cil. circle.	Gone, perhaps escaped through rupture.	Open .. ..
No. 421. James W., æt. 50. Blow on *L.* with fist 21 days ago. P.l. soon went, and severe pain came on lasting till excision. Lens displaced behind iris above, but in a.c. below. T. 3. Usual health good, but appears to have had gout.	Inflamed, early stage of sclerosis; there is some atrophy; thickness = ·52 mm.	Periphery applied ..	Access closed ..
No. 214. Joseph S., æt. 56. Blow 14 weeks ago, dislocating lens. T. n. then. Ten weeks ago the lens was removed and vitreous lost by an imperfect iridectomy upwards. Tension then came on. At excision T. 2. History more fully in vol. ix, p. 407.	Some atrophy (·54 mm.), moderate translucent substance among circular fibres. Some inflammation.	Periphery applied above, limb partly gone, stump in scar of iridectomy. Periphery free below.	Inflamed .. ..
No. 352. Mary W., æt. 38. Sclerotic perforated behind upper ciliary region by glass 5 months ago. No foreign body in eye. T. 1. History more fully in vol. ix, p. 397.	Upper part, some atrophy, thickness = ·55 mm. Some sclerosis of circular fibre region and of ciliary folds. Lower part better developed, = ·7 mm. Some sclerosis as above.	Periphery free ..	Well open .. ..

inflammatory disturbance has been rapidly followed by Glaucoma, on account of been enucleated.

Blood-vessels of ciliary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
Much enlarged. Walls extremely thin.	Central pit, like a physiological cup.	Neuritis, some translucent substance in sheath of central artery.	Walls seem neither hypertrophied nor hyaline.	Lens dislocated into vitreous chamber, but retains its upper and outer attachments.	Lig. pect. very thin, and considerably extended backwards.
Enlarged ..	A little swollen.	Neuritis, some translucent substance round new blood-vessels.	Walls seem neither hypertrophied nor hyaline.	Inflamed patches, in choroid like plastic.	Sclerosis in muscle much in advance of that in O.N.
Enlarged ..	Central physiological pit; margins much swollen.	Much neuritis, some translucent substance in sheath of central artery.	Walls hypertrophied.	Numerous hæmorrhages in retina.	The condition of the retinal b. vs. is very interesting in relation to the duration of the disease.
About normal.	Not cupped. Nuclei round the blood-vessels of papilla.	Neuritis ..	Nuclei round blood-vessels of retina, otherwise seem normal.	Many spots of disseminated choroiditis in a state of activity. Probably some enlargement of the supra-choroidal lymph-space as a clear gap of considerable size round long internal ciliary artery and nerve.	An exceptional case in many respects. Anterior part of retina cystic.
About twice as large above as below, where normal.	A central shallow pit. Sclerosis of sheaths of arteries of papilla.	No neuritis, bands of sclerosis not pressing much on nerve tissue.	Walls neither hypertrophied nor hyaline.	Choroid and Ret. destroyed at seat of wound by cicatricial tissue from sclerotic. Small post. synechiæ.	.. ..

GROUP 3.—*Eight cases in which a blow or wound attended by comparatively slight which the eye has*

History.	Ciliary muscle.	Iris.	Canal of Schlemm.	
No. 437. Joseph P., æt. 31. Blow on *R.* 8 months ago, after which sight faded. Especially painful last 14 days. T. 1. *L.* myopic 4 D.	Atrophied, inflamed, much sclerosed, but not very densely.	Periphery adherent. Pupil small, excluded, and occluded.	Access closed	..
No. 279. Isaac D., æt. 51. Blow on *R.* about 1 year ago, apparently followed by iritis. A second slight accident 11 months ago. P.l. rapidly went; and severe pain on *R.* side of head and in eye. At excision pupil irregular and excluded. A small opacity on cornea. T. 2.	Extreme atrophy and sclerosis.	Periphery adherent.	Access closed	..
No. 218. Thomas B., æt. 58. *L.* dim 4 years. Blow 4 weeks before excision. Remains of soft lens in a.c. Much pain. T. 2.	Atrophied. Some translucent substance just fibrillating.	Periphery adherent.	Access closed	..

GROUP 4.—*Twenty-two cases in which Increased*

A. In persons over 20

No. 83. Wm. K., æt. 57. *R.* vascular leucoma and staphyloma of cornea with entanglement of iris after perforating corneal ulcer 3 months ago. Now cornea leucomatous and prominent, T. +.	Atrophied; inflamed. Pigment cell layer of p.c.r. scattered and partly destroyed.	Inflamed; applied to posterior corneal surface, periphery adherent.	Access closed	..
No. 496. George K., æt. 47. *L.* corneal ulcer 5 months. Iris applied to cornea, and lens in contact with iris. T. full.	Of fair size, inflamed, early sclerosis.	Somewhat inflamed, periphery applied.	Access closed, inflamed.	

inflammatory disturbance has been rapidly followed by Glaucoma, on account of been enucleated.

Blood-vessels of ciliary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
Enlarged ..	..	Neuritis, especially on one side; much sclerosis of sheath of central artery.	Retina detached. Walls of blood-vessels as above.	Vitreous quite small, totally detached from posterior part of globe. Lens opaque *in situ*.	.. ..
Much enlarged.	Cupped ..	Decided vascular sclerosis.	Walls of blood-vessels as above.	.. ..	.. ..
Enlarged. Walls extremely thin.	Cupped ..	Neuritis and vascular sclerosis, apparently older than in muscle.	Walls of blood-vessels as above	.. ..	Glaucoma probably of a longer duration than 1 year.

Tension has followed a Perforating Corneal Ulcer.

years old. Eight cases.

Not enlarged.	Not cupped..	Neuritis and narrow bands of vascular sclerosis.	Walls neither hypertrophied nor hyaline.	.. ..	Herpes frontalis on *R.* side 13 years ago left then a small leucoma of cornea.
Not enlarged.	Central pit; early stage of sclerosis of walls of artery of papilla.	Neuritis and very early vascular sclerosis.	Walls neither hypertrophied nor hyaline.	.. ..	.. ..

GROUP 4.—*Twenty-two cases in which Increased*

A. In persons over 20

History.	Ciliary muscle.	Iris.	Canal of Schlemm.
No. 125. John H., æt. 60. Has had paralysis of *L.* seventh nerve for 12 years. Ulcer of *L.* cornea of 6 months' duration only. Cornea leucomatous and prominent. T. +.	Thin; inflamed clusters of nuclei in the connective tissue internal to muscle.	Adherent; atrophied.	Access closed, inflamed.
No. 18. John C., æt. 49. Perforating ulcer of *L.* cornea 7 months ago, said to be from exposure. Now cornea leucomatous and prominent.	Moderate atrophy; inflamed much.	Adherent; atrophied; also inflamed much near ulcer.	Access closed, inflamed.
No. 776. Jane R., æt. 60. Says that she had erysipelas of the face 9 months ago. After this got somewhat better; the *R.* eye came bad with a perforating ulcer. Cornea as above. T. 2.	Atrophied, much inflamed, some early sclerosis.	Adherent; atrophied; also inflamed much near ulcer.	Access closed, inflamed.
No. 287. Mary H., æt. 60. *L.* staphyloma of leucomatous cornea 1 year. T. +.	A little atrophied and inflamed; much sclerosis of circular fibres.	Adherent; atrophied; also inflamed much near ulcer.	Access closed, inflamed.
No. 181. Alfred M., æt. 50. *R.* leucoma of cornea from perforating ulcer 2 years ago. Bulging of thinned translucent upper corneal margin. T. +.	In *upper* ciliary region, total atrophy, some inflammation and much early sclerosis. In *lower;* musc. fair, early sclerosis of circ. fibres.	Base much inflamed, connective tissue uniting iris and cornea.	Access closed, inflamed.
No. 16. Wm. G., æt. 51. *L.* lost 2 years ago, by a perforating ulcer occurring, together with herpes frontalis. T. full. *R.* myopia = 3 D.	Moderate atrophy; inflamed.	Adherent and atrophied.	Access closed ..

Tension has followed a Perforating Corneal Ulcer,

years old. Eight cases.

Blood-vessels of ciliary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
Not enlarged.	Slight central depression, margin a little swollen.	Neuritis ..	Walls neither hypertrophied nor hyaline.	.. ..	.. ..
Not enlarged.	Mod. deep shelving pit.	Neuritis ..	Walls neither hypertrophied nor hyaline.	.. ..	.. ..
Not enlarged.	Mod. deep shelving pit.	Neuritis and early sclerosis.	Somewhat thick as if hypertrophied.	.. ..	.. ..
Not enlarged.	Mod. deep pit.	Neuritis and vascular sclerosis.	Somewhat thick as if hypertrophied.	.. ..	.. ..
Not enlarged.	Mod. deep pit.	Much neuritis and vascular sclerosis.	Walls neither hypertrophied nor hyaline.	.. ..	.. ..
Not enlarged.	Shallow depression.	Neuritis and early sclerosis.	Walls neither hypertrophied nor hyaline.	Fibres of intersheath space of nerve much hypertrophied.	Patient myopic. Herpes frontalis.

GROUP 4.—*Twenty-two cases in which Increased*

B. In persons under 20

History.	Ciliary muscle.	Iris.	Canal of Schlemm.
No. 463. Samuel L., æt. 4. *L.* staphyloma of leucomatous cornea from perforating ulcer 6 months ago. T. 1.	Not atrophied, inflamed.	One limb entangled in ulcer, atrophied and adherent; opposite limb inflamed, periphery adherent.	Access closed, inflamed, lumen very small.
No. 52. Henry M., æt. 7½ months. *R.* staphyloma of leucomatous thickened cornea from corneal ulcer, the result of ophthalmia neonatorum. T. +.	Much inflamed ..	Adherent, in places atrophied; much inflamed near ulcer.	Access closed, inflamed, lumen very small.
No. 428. Henry F., æt. 4. *L.* staphyloma corneæ from perforating ulcer 8 months ago. T. +.	Much inflamed ..	Adherent, in places atrophied, much inflamed near ulcer. Connective tissue on posterior surface of iris and of ciliary body.	Access closed, inflamed, lumen very small.
No. 146. William S., æt. 5. *L.* staphyloma corneæ from perforating ulcer 1 year ago. T. +.	Much inflamed ..	Adherent, atrophied, but in places inflamed.	Access closed, inflamed, lumen very small.
No. 363. George C., æt. 14. *R.* staphyloma corneæ from perforating ulcer 1½ years ago. T. +. *L.* myopia = 7 D.	Atrophied, inflamed, perhaps a little sclerosed.	Adherent, atrophied, but in places inflamed.	Access closed, inflamed, lumen very small.
No. 30. Joseph R., æt. 16. *L.* staphyloma corneæ from burn with molten lead 2 years ago.	Inflamed and a little atrophied.	Adherent and atrophied.	Access closed, inflamed, lumen very small.

Tension has followed a Perforating Corneal Ulcer.

Years old. Fourteen cases.

Blood-vessels of ciliary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
Not enlarged.	Central funnel-shaped pit.	Neuritis and early vascular sclerosis.	Walls neither hypertrophied nor hyaline.	Choroid contains occasional clusters of nuclei in its outer layers, and a general diffused increase of nuclei besides.	.. ..
Not enlarged.	Central funnel-shaped pit.	Much neuritis.	Walls neither hypertrophied nor hyaline.	Choroid has a general diffused increase of nuclei, its blood-vessels remain full of corpuscles.	.. ..
A trifle large.	Central funnel-shaped pit.	Neuritis and early vascular sclerosis.	Walls perhaps somewhat hypertrophied. Some episcleritis.	Choroid has a general diffused increase of nuclei, its blood-vessels remain full of corpuscles.	.. ..
Not enlarged.	,, ..	Neuritis ..	Walls neither hypertrophied nor hyaline.	Choroid has general diffused increase of nuclei, and blood-vessels full of corpuscles.	.. ..
Enlarged ..	Central deep pit; dense sclerosis of walls of blood-vessels of papilla.	Neuritis and vascular sclerosis.	Walls hypertrophied.	Choroid has general diffused increase of nuclei, and blood-vessels full of corpuscles. A layer containing irregular scattered pigment exists on the internal surface of ciliary body.	.. ..
Not enlarged.	(?)	Neuritis ..	Walls neither hypertrophied nor hyaline.	Choroid has general diffused increase of nuclei.	.. ..

GROUP 4.—*Twenty-two cases in which Increased*

B. In persons under 20

History.	Ciliary muscle.	Iris.	Canal of Schlemm.
No. 376. Keziah M., æt. 15. *R.* staphyloma after corneal ulcer 4 years ago. T. 3 before an iridectomy was done recently. At excision, T. n. Globe removed on account of pain.	Inflamed, perhaps a little sclerosis of region of circular fibres.	*One limb* in ulcer, *other* inflamed, periphery adherent and atrophied.	Access closed, inflamed, lumen very small.
No. 175. Elizabeth F., æt. 16. *R.* staphyloma corneæ from ulcer in ophthalmia neonatorum. Globe much enlarged. T. +.	Somewhat inflamed, a little atrophied.	Adherent, atrophied mostly, but inflamed in places.	Access closed, inflamed, lumen very small.
No. 109. Arthur L., æt. 12. *R.* staphyloma corneæ from ulcer in ophthalmia neonatorum. Globe much enlarged. T. +.	Much inflamed, stretched, and atrophied; thin irregular layer containing pigment on the internal surface of the ciliary body.	Adherent and atrophied.	Access closed, inflamed, lumen very small.
No. 347. Thomas R., æt. 12. *R.* staphyloma corneæ from ulcer in ophthalmia neonatorum. Globe much enlarged. T. +.	*Upper ciliary region*, extreme atrophy; some inflammation; no sclerosis. *Lower*, inflamed; mod. atrophy; abundant connective layer, containing pigment on inner surface of ciliary body.	Adherent and atrophied.	Access closed, inflamed, lumen very small.
No. 34. Sarah W., æt. 13. *R.* staphyloma corneæ from ulcer in ophthalmia neonatorum. Globe much enlarged. T. n. now.	Moderately atrophied and much inflamed; a thin layer of newly formed nuclear bodies on internal surface of ciliary body.	Mostly adherent and atrophied, especially at base; limb where free inflamed.	Access closed, inflamed, lumen very small.

Tension has followed a Perforating Corneal Ulcer.

ears old. Fourteen cases.

lood-vessels of iliary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
erhaps a trifle enlarged; walls of good thickness.	Central pit, edges swollen.	Neuritis (?) (nerve very short in specimen.)	Walls neither hypertrophied nor hyaline.	Choroid has general diffused increase of nuclei.	.. ..
ot enlarged.	Cupped ..	Neuritis and vascular sclerosis; numerous new blood-vessels.	Walls neither hypertrophied nor hyaline.	Choroid has general diffused increase of nuclei.	Anterior part of retina is very cystic.
ot enlarged.	Cupped ..	Neuritis and vascular sclerosis; numerous new blood-vessels.	Walls neither hypertrophied nor hyaline.	Choroid has general diffused increase of nuclei, and is œdematous.	.. ..
omewhat enlarged, but walls of fair thickness.	Central pit. lam. crib. very concave; concavity nearly filled by nuclear tissue; margins of papilla swollen.	Dense vascular sclerosis.	Walls hypertrophied; lumen very small, or wanting; black pigment often in or round their wall.	Abundant nuclei in ch. and in adherent retina. Pigment epithelium layer destroyed, its pigment black and irregularly scattered.	Suspicion of hereditary syphilitic taint.
ot enlarged.	Fairly deep, universal cup.	Much neuritis.	Walls neither hypertrophied nor hyaline.	.. ..	.. ..

GROUP 4.—*Twenty-two cases in which Increased*

B. In persons under 20

History.	Ciliary muscle.	Iris.	Canal of Schlemm.
No. 38. Edward P., æt. 14. *R.* staphyloma corneæ from ulcer in ophthalmia neonatorum. Globe much enlarged. T. formerly increased, now − T. 3.	Inflamed, and containing between its bundles of fibres numerous red blood corpuscles, no sclerosis; layer on ciliary body, as above.	Mostly adherent and atrophied, especially at base; limb where free inflamed.	Access closed, inflamed, lumen very small.
No. 468. Rebecca R., æt. 16½. *R.* cornea flat, leucomatous. Globe enlarged in antero-posterior and lateral diameters, and T. + from corneal ulcer when 7 weeks old. Some nystagmus of *both*. *L.* myopic about 7·5 D.	Atrophied and much inflamed; layer on ciliary body, as above.	Mostly adherent and atrophied, especially at base; limb where free inflamed.	Access closed, inflamed, lumen very small.
No. 224. Emma M., æt. 19. Dense leucoma of cornea from ulcer in infancy. Ciliary region of sclerotic, and cornea thin and staphylomatous. Globe enlarged.	Almost total atrophy, some inflammation; connective layer on internal surface of ciliary body.	Adherent and extremely atrophied.	Access closed, inflamed, lumen very small.

GROUP 5.—*Five cases in which the anterior part of the Globe is especially enlarged Traumatic or Ulcerative Origin, and whether with*

No. 123. Mary H., æt. 7½. Congenital buphthalmos of both eyes. T. 2. Iridectomy attempted, followed by hæmorrhage into cavity of vitreous and under choroid. Immediate excision.	Atrophied; inflamed, fibres separated by much hæmorrhage.	Atrophied, and adherent to cornea.	Closed .. ..

Tension has followed a Perforating Corneal Ulcer.

ears old. Fourteen cases.

lood-vessels of iary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
ot enlarged.	Fairly deep, universal cup.	Much neuritis.	Walls neither hypertrophied nor hyaline.	Choroid has diffused nuclear increase; blood-vessels full.	.. ..
ot enlarged.	Not cupped, appearances characteristic of myopia.	A little neuritis; some translucent substance round smaller blood-vessels; numerous vessels of new formation in walls of central artery.	Walls neither hypertrophied nor hyaline.	Choroid contains patches of dense nuclear bodies in neighbourhood of macula. A little episcleritis.	Globe larger in lateral than in vertical diameter.
ot enlarged.	Deep, rounded cup.	Neuritis and much sclerosis.	Walls neither hypertrophied nor hyaline.	Choroid has diffused nuclear increase, and blood-vessels full.	.. ..

n consequence of Increased Tension from Disease occurring in Infancy, whether of Anterior Synechia or with deep Anterior Chamber.

ot enlarged.	Central pit ..	Neuritis and vascular sclerosis.	Walls of capillaries of int. granular layer much thickened and hyaline.	Ch. contains diffused nuclear increase, and full blood-vessels. It is thickened.	Patient is in all probability syphilitic.

GROUP 5.—*Five cases in which the anterior part of the Globe is especially enlarged Traumatic or Ulcerative Origin, and whether with*

History.	Ciliary muscle.	Iris.	Canal of Schlemm.
No. 69. Constant C., æt. 12. Buphthalmos from penetrating wound 8 years ago. + T.	Much inflamed and somewhat atrophied.	Atrophied, and adherent to cornea.	Closed .. ..
No. 787. Arthur M., æt. 10. Buphthalmos from blow in infancy. Pupil occluded and excluded. Universal post. synechiæ. A.c. extremely deep.	Much inflamed ..	Universal posterior synechia.	(?)
No. 599. Ann L., æt. 22. Buphthalmos and marginal corneal staphyloma above from wound when æt. 5. + T. at excision.	Atrophied, more especially above, and moderately inflamed.	Atrophied, and adherent to cornea.	Access closed ..
No. 760. John A., æt. 54. *R.* buphthalmos from ulcer when æt. 1. T. n. now. *L.* V = $\frac{20}{20}$.	*Above*, atrophied much, and slightly inflamed. *Below*, much inflammation and atrophy.	Atrophied, and adherent to cornea.	Access closed ..

in consequence of Increased Tension from Disease occurring in Infancy, whether of Anterior Synechia or with deep Anterior Chamber.

Blood-vessels of ciliary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
Not enlarged.	Deep central cup.	Much neuritis and early vascular sclerosis.	Walls of arteries much thickened.	Ch. contains diffused nuclear increase, and full blood-vessels. It is thickened.	.. ..
A little enlarged.	Retina detached.	Neuritis and some vascular sclerosis.	Walls neither hypertrophied nor hyaline.	Choroid near o. d. contains considerable excess of nuclear bodies, but its innermost stratum is apparently converted into connective tissue.	.. ..
Enlarged ..	Deep cup ..	Neuritis and decided vascular sclerosis.	Apparently thickened.	Ch. contains diffuse nuclear increase.	.. ..
Enlarged ..	Shallow shelving cup.	Neuritis and much vascular sclerosis. Much sclerosis of walls of arteries of papilla, nearly obliterating their lumen.	Walls of arteries immensely thickened and translucent, containing a few nuclei. In other places, where there is abundant choroido-retinitis, the pig. epith. is destroyed, the pig. black and irregularly scattered. Some is found round the blood-vessels of which the lumen is sometimes obliterated.	.. ..	.. ..

GROUP 6.—*Three cases of Glaucoma*

History.	Ciliary muscle.	Iris.	Canal of Schlemm.
No. 509. James F., æt. 42. *L.* sight very dim 3 months ago. Now it presents distinct iritis serosa, not at all severe. *R.* a *little* dim 3 months ago. Iridectomy was done on *this* eye then. *R.* at *excision;* much swelling at operation wound. T. 2. Dots on posterior corneal surface, &c.	Not atrophied, much inflamed.	Periphery free, except at operation wound. Iris is much inflamed.	Much inflamed. Access not closed by iris, except near operation wound.
No. 285. Sarah F. *L.*, cataract 1 year. Inflamed 6 months. T. +. Iridectomy 5 months. At *excision L.* much lymph in pupil. *R.* operated on for cataract 1½ years ago. Now moderate vision.	Not atrophied, much inflamed. Apparently some sclerosis of internal part of ciliary body.	Periphery widely free.	Inflamed .. ..
No. 274. Elizabeth H. *R.* dim 3 years. Now T. +. Appearances typical of what is known as iritis serosa. *L.* about normal.	Not atrophied, much inflamed, no sclerosis.	Periphery widely free.	Inflamed .. ..

e to "Iritis Serosa," &c.

ood-vessels of ary region.	Optic disc.	Optic nerve.	Retinal blood-vessels.	Other pathological conditions.	Remarks.
t enlarged.	Central pit ..	Considerable neuritis.	.. ..	Much episcleritis. There are cells on the posterior surface of cornea. Choroid much inflamed.	The iridectomy was not done at Moorfields Hospital.
t enlarged.	Not cupped .	Some neuritis.	.. ..	Much episcleritis. There are cells on the posterior surface of cornea. Choroid much inflamed.	.. ..
t enlarged.	Not cupped .	Some neuritis.	.. ..	Much episcleritis. There are cells on the posterior surface of cornea. Choroid much inflamed.	.. ..

EXPLANATION OF FIGURES IN CURATORS' PATHOLOGICAL REPORT.

PLATE I.

FIG. 1.—Female, æt. 40. To illustrate the normal anatomy of the parts. Death from blood-poisoning.

FIG. 2.—Case 274 in Curator's Report (see page 128). Increased tension probably of long standing. Four months under observation in the Out-patient's Department.

FIG. 3.—Case 101 (see page 114). Increased tension of less than 14 days' duration.

FIG. 4.—Case 483 (see page 106).

PLATE II.

FIG. 5.—Case 401 (see page 96).

FIG. 6.—Case 452 (see page 104). In this case there were marked inflammatory symptoms, and the aqueous chamber at its centre was deeper than normal.

FIG. 7.—Case 395 (see page 94).

Fig. 8.—Case 338 in Curator's Register. A young man, æt. 19, had been when two years old the subject of a perforated corneal ulcer. The eye became apparently the subject of some secondary increase of tension. *At excision*, it is enlarged especially in an antero-posterior direction, and its posterior segment is the subject of a staphylomatous thinning. There is slight increase of connective tissue in the nerve, but nothing comparable with that seen in the next figure.

FIG. 9.—Case 340 (see page 102). It shows in transverse section the typical condition of optic nerve found in the glaucoma of adults and old persons, described in the Report as Vascular Sclerosis.

Fig 1. ×15.

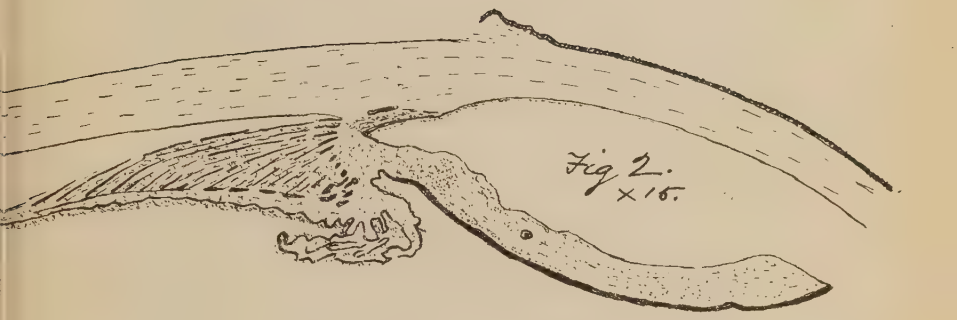

Fig 2. ×15.

Fig 3. ×15.

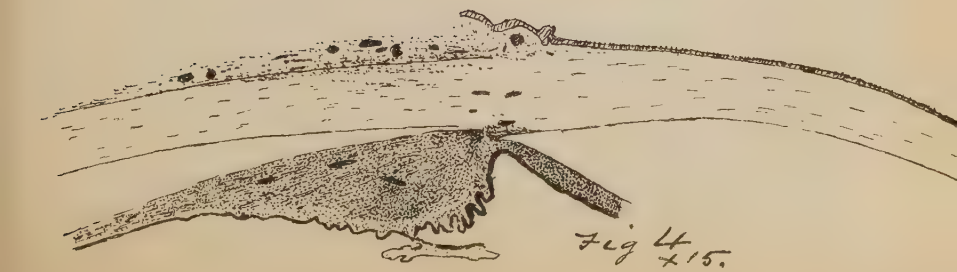

Fig 4. ×15.

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Fig 5. ×15.

Fig 6. ×15.

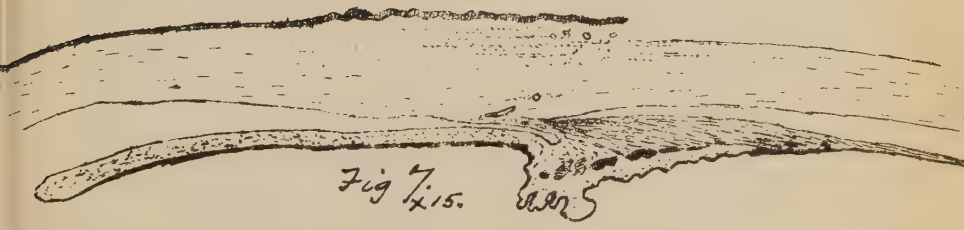

Fig 7. ×15.

Fig 8 ×20

Fig 9. ×20.

Fig 10. (×55)

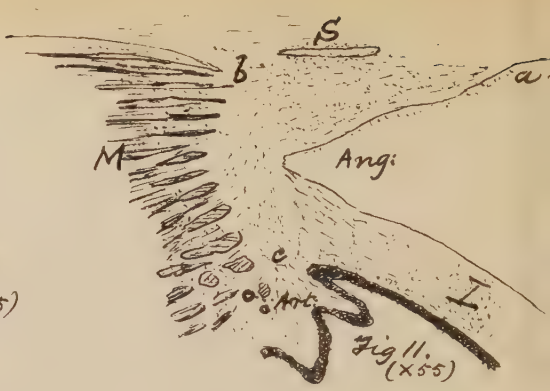

Fig 11. (×55)

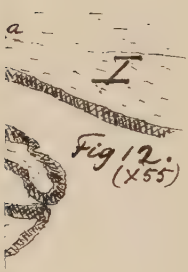

Fig 12. (×55)

Fig 13. (×55.)

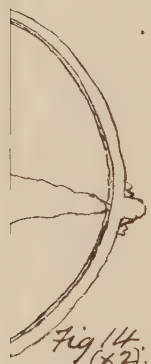
Fig 14. (×2).

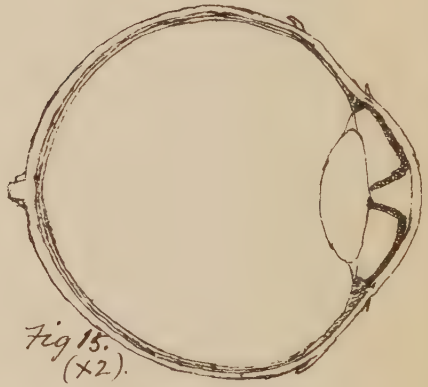
Fig 15. (×2).

Fig 16. (×2).

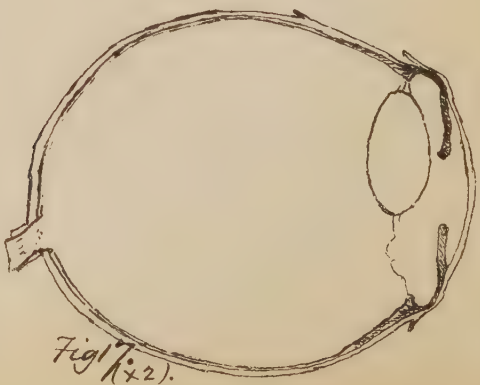
Fig 17. (×2).

Plate III.—Explanation of diagrams to show the change in shape of the Ligamentum Pectinatum, which is the principal cause of the approximation of the periphery of the iris to the cornea in glaucoma.

The part specially affected is that named by Gerlach the Ligamentum Annulare (see Gerlach "Beiträge zur normalen Anatomie des Menschlichen Auges." Leipzig, 1880). The most anterior stratum of this by its connexion on the one hand with Descemet's membrane, and on the other with the connective tissue framework of the iris narrows the peripheral part of the anterior chamber of the Aqueous. The more posterior and thicker stratum by *its* diminution in size contracts together the fibres of origin of the meridional part of the ciliary muscle. The terms here used are adopted from Gerlach :—

a = inner angle.	S. = Canal of Schlemm.
b = outer angle (usually rounded).	Art. = Circulus Arteriosus.
c = posterior angle (usually acute).	M. = Meridional part of ciliary muscle.
bc = outer border.	Ang. = Angle of Anterior Chamber of Aqueous.
I. = Iris.	

Figs. 10, 11, 12, and 13, are enlarged representations of the Ligamenta Pectinata of Figs. 1, 2, 5 (reversed), and 6 respectively.

Appendix to Curator's Report.

Since the above report has been in the press four eyes with increased tension have been examined immediately after excision after being frozen.

The eyes were divided vertically in an antero-posterior plane, cutting from above downwards.

The sections are represented in the accompanying figures.

Fig. 14.—John McD., æt 57. 30 years ago he had an accident with gunpowder, after which black particles remained imbedded in his left cornea. 12 months ago he could still read large print, though his eye had become considerably myopic. Three weeks ago it became painful and a little red: a few days later he accidentally discovered it had only p.l. At excision T 1½. Measurements of *globe:* Antero-posterior 25 mm., vertical 23·5 mm. *Lens* diameter 9·3 mm. Antero-posterior 4·75 mm. Depth of a.c. from posterior corneal surface 2 mm.

Fig. 15.—Thomas I., æt. 19. Vision lost by iritis three years ago. At excision T 2. Pupil excluded and occluded. *Globe:* Antero-posterior 25·5 mm., vert. 24·75 mm. *Lens* diam. 8·75. Antero-posterior 2·75 mm.

Fig. 16.—James Buckland, æt. 59. Seven months ago his eye became inflamed and painful, with small corneal infiltrations and hypopyon; the pupil was inactive with posterior synechiæ. T + : V = p. l. One month later iridectomy upwards was done. In another month the extensively infiltrated cornea was incised by Sæmisch's method; since then severe pain till excision. The a.c. is said to have reformed previous to excision. T+. *Globe:* Antero-posterior 25 mm., vert. 24 mm. *Lens* diam. 11 mm. Antero-posterior 5·5 mm. Abundant soft material immediately inside lens capsule.

Fig. 17.—Thomas C., æt. 3. Glioma with increased tension. The posterior segment of the globe full of glioma matter, but the aqueous chamber is as yet quite free. It is much increased in size.

ON SOME CHANGES WHICH OCCUR IN THE BLOOD-VESSELS IN DISEASES OF THE EYE, CONSIDERED IN THEIR RELATION TO GENERAL PATHOLOGY.

By Walter Edmunds, M.D., and W. A. Brailey, M.D.

The structure of the eye in comparison with its size is very complex; in it are found representatives of the chief tissues of the body; and as the eye, in addition to numerous local affections, is frequently involved in general diseases, a collection of diseased eyes will afford a rich field for pathological study.

Such a collection there is in the museum of Moorfields Hospital, where microscopical preparations of a very large number of eyes are preserved, the specimens being particularly easy of comparison from their having all been made in the same manner, namely, hardened in chromates, stained in logwood, and mounted in Dammar. The following observations are based on the study of this collection.

The thickening of the coats of the arteries which occurs throughout the body in some form of Bright's disease was pointed out many years ago by Dr. George Johnson, and the discussion which subsequently arose between him, Sir W. Gull, and Dr. Sutton, as to the exact nature and interpretation of the change, again directed attention to this hypertrophy, as to the existence of which there is no question whatever.

In Syphilis, too, hypertrophy of the walls of arteries has been described by Heubner in Germany, and in this country specimens of the disease have from time to time been exhibited at the Pathological Society, more especially when the memorable discussion on Syphilis was being held.*

Widespread arterial change has also been described in

* *See* Pathological Society's Transactions for 1877, where also will be found an account of Dr. Klein's "Observations on the Arterial Changes in Scarlet and Typhoid Fevers."

Diabetes, and an interesting example of this will be found fully related in the last volume of these Reports by Dr. Stephen Mackenzie.

It must not, however, be inferred from these observations that arterial hypertrophy is necessarily a general or widespread disease: for, as we shall endeavour to show, it sometimes is found in the eye alone: the thickened arteries, which have often been described in Retinitis Albuminurica, are part no doubt of a general disease; but a similar condition of vessel is found in Glaucoma, which, at all events in the cases in which it is secondary to injuries, appears to be a purely local affection.

Before describing the changes the blood-vessels undergo in Glaucoma, it is necessary to point out that under this term we include all cases in which the intraocular tension is increased, whether primarily, as in Glaucoma strictly so called, or secondarily, as in Glaucoma supervening on injury, corneal ulcer, or inflammation of the uveal tract. Under this head, too, we include the hæmorrhagic Glaucomas, and the condition known as Buphthalmos, the peculiar appearance to which this name is applied being due merely to the action of increased tension in children in whom the cornea, where not leucomatous, readily stretches.

In another part of these Reports will be found analyses of the morbid appearances in 88 cases of Glaucoma (see page 94): and among the 23 cases of the non-hæmorrhagic form in which the retina was carefully examined, the retinal arteries were distinctly hypertrophied in eight. The proportion is very different in the hæmorrhagic form of the disease (Group II), for out of the 17 cases in which the retina was examined, the arteries were found thickened in all but one. This is not due to any radical difference in the nature of the two forms of the disease, but merely to the fact that hæmorrhages only occur in the more severe or advanced cases, and that consequently only these are included in the hæmorrhagic category.

Group III is composed of cases of comparatively slight

injury to the eye in which it has become necessary to perform excision within a year on account of Secondary Glaucoma. Among these the retinal arteries were only found to be hypertrophied in one case, the history of which is of special interest.

A man, 50 years of age, received a blow on the eye with the fist; he had severe pain almost immediately, with tension becoming greatly increased (T. 3) for three weeks when the globe was excised. On examining the eye, there were found amongst other changes numerous capillary hæmorrhages in the retina, the arteries of which were distinctly hypertrophied. The patient was not the subject of any general disease, nor is there any reason to think that the eye was in any way abnormal before the injury, and if this were the case, the hypertrophy of the arteries must have come on in 21 days.

Among the 22 cases of Glaucoma following perforating ulcers (Group IV), thickened retinal arteries were found in five cases.

Lastly, among the five cases of Buphthalmos (Group V), the same change was seen in three, in one of which the diseased condition of blood-vessels was very manifest, for, while the operation of iridectomy was being performed, the whole globe filled with blood, and it became necessary to excise forthwith.

Thus we see that in a considerable proportion of cases of Glaucoma, the retinal arteries are hypertrophied. The thickened vessels in a later stage contract, and their lumen becomes smaller: in two cases they were impervious, and reduced to the condition of fibrous cords: it may be added that in Glaucoma the vessels never present any proliferation of their endothelium.

In cases in which there has been choroido-retinitis the arteries and their sheaths are frequently found impregnated with pigment molecules, which have apparently come from the diseased pigment epithelium layer of the retina.

The pigment also surrounds the aneurysmal dilatations which these arteries frequently exhibit.*

But the arteries are not the only vessels which are affected: the capillaries often have their walls thickened and composed of a structureless, highly refracting substance, which we have called "hyaline;" they gape on section, and are often aneurysmal. This last condition may be seen either in section in which a capillary happens to be exposed in its length, or by looking at a portion of the retina on the flat.† The retinal veins are not nearly so conspicuously, if at all, affected.

The central artery of the optic nerve is also altered, but the changes it undergoes are of a different character.

In all cases of Glaucoma the optic nerve is found inflamed or sclerosed; and in this inflammation (for sclerosis is probably only a late stage of neuritis) the central artery participates as it passes through the nerve. The only change noticed in the earliest cases is an enlargement of its vasa-vasorum and the appearance of nucleated bodies in its lymph-sheath, that is to say, in the loose tissue which forms part of its adventitia. In the next stage a structureless substance, in which nuclei lie sparsely imbedded, is seen in the lymph-space; and in the later stage this substance becomes fibrillated, and finally converted into a dense fibrous tissue in which no nuclei can be detected.

Some evidence that this is the order in which the changes occur is obtained from a comparison of the duration of the Glaucoma in the cases of Group III.

The last two stages (the translucent and the fibrous) we conceive to be the conditions of artery which Gull and Sutton have included under the name "hyalin-fibroid."

Identical changes have been described in the arteries of

* *See* Poncet (de Cluny), Atlas, pl. LI (1879).

† Retinal aneurisms were first described by Liouville: he found them occurring as part of a general condition which he considered as an aneurysmal diathesis; no increase of ocular tension is mentioned in any of his cases. (Comptes rendus de la Société de Biologie, 1868.)

sclerosed portions of the brain and spinal cord (as we ascertained subsequently to observing them) by Charcot.*

As to the causation of these vascular changes the starting point seems to be the inflammation (or sclerosis) which, as we have said, is found in all cases of Glaucoma. This inflammation affects the central artery in its course through the optic nerve, and thus occasions the changes in its lymph-sheath: the inflammation is propagated forwards to the retinal arteries, but in them it cannot take quite the same form, for they have not (as far as our observations go) lymph-sheaths.

In the retinal arteries it is difficult to define the exact seat of origin of the inflammatory changes, but in the central artery of the nerve it is the muscular coat which appears most often involved.

The varicose condition of the capillaries might, perhaps, be explained by the fact that the thickened and probably inelastic walls of the retinal arteries would more directly transmit the pressure of the blood, which might then be excessive for the altered walls of the capillaries.

If the disease of the retinal arteries is due to the central artery being affected in its course through the optic nerve, it will follow that the choroidal arteries, which have an entirely distinct origin, will not be affected, and, in fact, they are not, at least in the great majority of cases. Indeed, in nearly all the specimens the contrast between the thickened retinal arteries and the natural choroidal vessels is very marked, and quite confirms our proposition that the arterial change is strictly local.

One other vascular change in Glaucoma still remains to be mentioned: the arteries in the ciliary region are often greatly dilated. This change may be well seen in the "Circulus arteriosus iridis major," which is frequently found vastly enlarged, with it walls even more than proportionately thinned.

Thus we see that in Glaucoma there are frequently found in the same eye hypertrophied retinal arteries, those of the

* "Leçons sur les Maladies du Système Nerveux," pp. 181, 1872.

choroid unaltered, and the arteries of the ciliary region dilated and thinned; a condition which is not to be explained by any general arterial disease.

In syphilis the retinal arteries are affected, but in a different manner. Their walls on transverse section are seen dissociated, and thicker than natural, the thickening being more than can be accounted for by the dissociation of the fibres of which they are composed. That this change is probably in the first place inflammatory may be inferred from the frequent presence of staining nuclei in the dissociated walls. It may be a later stage of the syphilitic arteries described by Barlow and others at the Pathological Society. Their appearances closely resemble those observed by Heubner in the intima, and figured in his mongraph (1874).

As to the central artery of the optic nerve, although no doubt its sheath can participate in the results of surrounding inflammation, yet this vessel appears to be unaffected when syphilis is uncomplicated by any other diseased condition.

In syphilis, as in Glaucoma, a varicose condition of the capillaries is sometimes found: also, when the retina is implicated in a choroido-retinitis, the retinal arteries may become pigmented.

In syphilis the choroidal arteries do not appear to be affected, and those of the retina present the same characters, whether the disease be acquired or inherited.

In discussing the distinctly inflammatory diseases of the eye it will be convenient to consider more especially the serous inflammation of the uveal tract, as in this the changes are slower and can be more readily determined.

In this there are two distinct types of arterial disease: one may be observed in the choroid, the other in the retina, which is always affected secondarily and to a less degree.

The choroidal arteries participate in the general character of the choroidal inflammation, which is essentially a proliferation of the fixed corpuscular elements of its stroma.

In the more advanced forms of this inflammation the endothelium of the arteries participates extensively, whereas

the wall presents comparatively but few corpuscular elements. In the iris the smallest arteries are often seen completely blocked by this endothelial proliferation, while the thick, nearly pellucid walls, which characterise them in this structure, are nearly free from staining elements, though they are closely surrounded by the crowded corpuscular elements of the inflamed part; but in the retina, which is always involved to a minor degree in the inflammation, the change is not one of connective-tissue proliferation, but chiefly of diapedesis.

Transverse sections of the retinal arteries show their walls crowded by leucocytes, and thus greatly thickened; their lumen is reduced by a narrow chink, not by endothelial increase, but by the swelling of the thickened walls. On the vitreous surface of the hyaloid, corresponding to the retinal arteries, are leucocytes which have migrated from these vessels.

In the same way it is the connective tissue of the optic nerve which is chiefly affected, for the nuclear bodies which are found in it lie not so much in the loose tissue of the lymph-sheath of the artery as in the dense connective tissue external to it, which is continuous on its outer aspect with the fibrous trabeculæ of the nerve. In conformity with this distribution the nuclei are seen extending horizontally in the framework of the nerve for some little distance. In cases of descending neuritis, on the other hand (the choked disc of authors), we have found leucocytes crowding the loose tissue of the sheath of the artery, the fibrous stroma of the nerve being comparatively free from them.

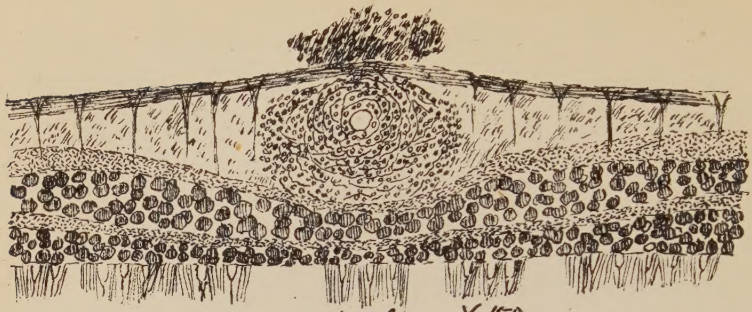

Fig A X 150

Fig B x 150

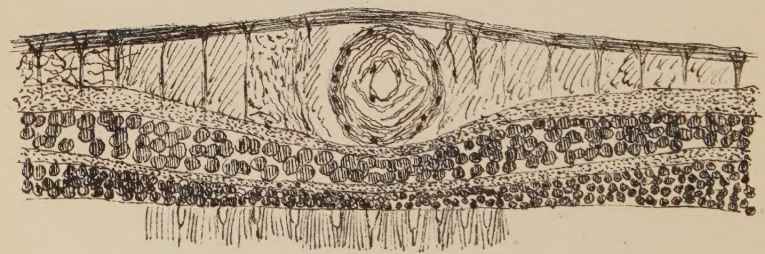

Fig C x 150

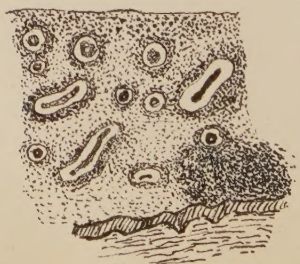

Fig D x 20

EXPLANATION OF DIAGRAMS.

FIG. A is the Retina in Case 274, mentioned in the Curator's Report, Group 6. It was a typical case of Iritis Serosa; the eye had been failing for three years.

FIG. B. A typical case of Hæmorrhagic Glaucoma, No. 500 in Curator's Register.

FIG. C. Retinal Artery in a case of Hereditary Syphilis, No. 187 in Curator's Register. This case will be found reported in Mr. Hutchinson's work on Syphilitic Diseases of the Eye and Ear, p. 179.

FIG. D. To show condition of Blood-vessels of Iris in a case of sympathetic inflammation, No. 396 in Curator's Register.